6

Human Health Risk Assessment: Lead Exposure and Uptake—Use of the IEUBK Model

MODEL DEVELOPMENT BACKGROUND

Childhood Lead Exposure and Model Development Needs

Lead exhibits a broad range of toxic effects on animal systems, organs, and cellular biochemical and metabolic processes. A National Research Council report (NRC 1993) titled *Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations* concluded that "lead causes nonspecific, decremental loss of tissue and organ function, with no important pathognomonic manifestations of toxicity." Furthermore, exposure to lead occurs by multiple pathways and routes. Because many environmental reservoirs are contaminated with lead, it is seldom possible to identify a sole significant source of lead exposure.

A primary human exposure pathway to lead is through soil and dust, which children are assumed to incidentally or deliberately ingest. Empirical evidence for this assumption comes from reports of excess amounts of soil tracer elements, especially silicon and aluminum, in the feces of children (Wong et al. 1988; Calabrese et al. 1989; Davis et al. 1990). However, because of the inherent difficulties associated with sampling feces from many children over long periods, available data are limited. As a consequence, actual rates of soil ingestion are somewhat uncertain. Quantitative evidence of hand-to-mouth activity in children has been produced by videography (Zartarian et al. 1997; Reed et al. 1999; Freeman et al. 2001). It is also well established that some fraction of the lead found in soils is

absorbable in mammalian gastrointestinal tracts (Casteel et al. 1996a-d, 1997a,b, 1998a-e). Studies generally are consistent in demonstrating that a nonnegligible fraction of lead in soil can be absorbed but that the efficiency of absorption depends on multiple factors including chemical speciation of lead, other dietary components, and particle size of soil ingested. Typically paint-derived lead is relatively available for absorption, whereas lead associated with sulfide minerals is relatively unavailable.

Under the environmental health paradigm, preventing injury is the first choice (see Box 6-1). As discussed in Chapter 5, the primary threat presented by lead relates to its ability to cause developmental deficits in children. Although chelation therapy can be applied to reduce body burdens of lead, available information suggests that chelation is not effective in restoring neurological function (Rogan et al. 2001). Hence a "monitor and react" strategy, even if conducted well, cannot prevent injury. The primary prevention strategy (Campbell and Osterhoudt 2000; Rosen and Mushak 2001) is widely recognized as the only truly effective method for eliminating pediatric lead poisoning; this requires a degree of predictive capability for both risk assessment and risk management.

The U.S. Environmental Protection Agency (EPA) has adopted a strategy that entails modeling lead exposure rather than biomonitoring as the first line of defense. Existing epidemiological evidence for health effects of lead exposure is anchored to BLLs rather than to dose rates. The relationship between dose and blood level is complicated by the fact that lead is stored in bone. This entails a greater level of modeling sophistication than the standard risk assessment guidance for Superfund (RAGS) paradigm.

A primary difference between lead risk assessment and cancer and noncancer risk assessment for other chemicals or compounds is that BLLs can be readily measured in individuals and used to "ground-truth" risk calculations. BLLs provide an integrated picture of lead exposure over the preceding months to years, depending on age and other characteristics of

BOX 6-1 Preventing Lead Exposure

Children with access to lead-contaminated soils are likely to be exposed to that lead. To establish levels of lead contamination that would not be expected to present unacceptable or unavoidable risk, it is necessary to define the relationship between magnitude of exposure and level of soil contamination.

Children exposed to lead who develop elevated blood lead levels (BLLs) may have already been irreversibly damaged by the time they have been identified in screening programs. A primary prevention strategy requires the predictive capability of models for exposure risk assessment and management activities.

exposure. In addition, a large body of research exists linking levels of lead in blood to various health effects. As a result, the toxicity and risk characterization steps of a typical risk assessment, as described in the previous chapter, are combined in lead risk assessment into a prediction of BLLs arising from associated lead exposures. Whether risk is deemed acceptable or unacceptable is assessed by comparing the predicted BLLs with target BLLs established by the Centers for Disease Control and Prevention (CDC 1991) and adopted by EPA.

EPA uses two predictive blood lead models for risk assessment purposes: the IEUBK model for children up to the age of 7 years (84 months) and the adult lead model for adolescents and adults. In this chapter, we discuss only the integrated exposure uptake biokinetic (IEUBK) model because children are the most susceptible population and residential soil lead cleanup levels generally are set on the basis of childhood lead risk.

Predictive Blood Lead Models

Lead exhibits a broad range of toxic mechanisms across a variety of target organ systems, and because it has multimedia exposure pathways, the overall dose-response relationships for lead are more complex than those of some other toxic agents. This argues for both biokinetic and pharmacokinetic methods of study to elucidate the concentration and rates of change of lead in various body reservoirs. Mathematical models are particularly useful in this regard because the impacts of lead exposure need to be established on a population-wide basis (NRC 1993). Thus, a variety of predictive blood lead models have evolved for use in lead exposure risk assessment and risk management activities.

Two kinds of model development approaches can be used for predicting blood lead values in response to environmental exposure factors. Slope factor models propose a simple linear relationship between BLL and the uptake or intake of lead from environmental media (air, water, food, soil, dust). If uptake is modeled, in contrast to lead intake, the models are sometimes referred to as biokinetic slope factor models. Examples include those developed by Carlisle and Wade (1992), Bowers et al. (1994), Stern (1994, 1996), the Ontario Ministry of Energy and Environment (OMOEE) (1994), and the Agency for Toxic Substances and Disease Registry (ATSDR 1999). The comparative functioning of several of these models and the multicompartment models described below are detailed in a review of adult lead models examined by the technical review workgroup for lead (TRW) (EPA 2001a).

Multicompartment predictive blood lead models simulate the movement and concentration of lead in several interconnected tissue compartments with blood or extracellular fluid (plasma) serving as the exchange

medium. Rabinowitz (1998) reviewed the early development of this approach, illustrating the usefulness of such models after the experimental application of radioactive tracers showed the relatively short half-life of lead in blood (about 1 month) compared with a 15- to 20-year residence time in skeletal tissue. Models of this type have been developed by Rabinowitz et al. (1976), Marcus (1985), Bert et al. (1989), O'Flaherty (1993), Leggett (1993), and EPA (1994a,b). A simple depiction of a multicompartment model, similar to that of Rabinowitz et al. (1976) is shown in Figure 6-1. Biokinetic and pharmacokinetic models relate exposure dose to the lead concentration in various target tissues; they represent the mathematics of the time course of absorption, distribution, metabolism, and excretion (ADME) of the substance being followed. Biological, physiological, and physicochemical factors all influence the rate and extent of ADME.

Several mathematical approaches underlie the pharmacobiokinetic (PBK) model structures: in diffusion-limited models, such as the IEUBK model, rates of change of lead concentration in the various compartments are defined by the rates of transfer across compartment boundaries. The time parameter is represented in the diffusion rate constants. Lead transfers are typically assumed to follow first-order kinetics; exchanges are repre-

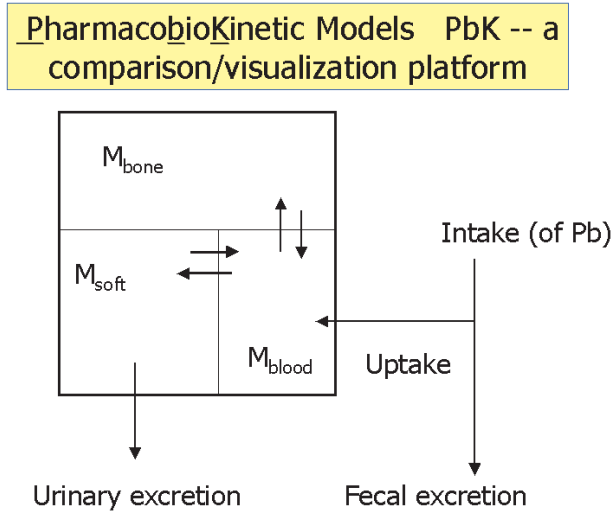

FIGURE 6-1 Simple model framework illustrating compartments and pathways of exchange for a pharmacobiokinetic model of lead in the human system. SOURCE: Rabinowitz et al. 1976. Reprinted with permission from the American Society for Clinical Investigation.

sented by first-order rate constants. However, such "constants," may take on age-specific values, an important characteristic of PBK models applied to children's lead exposure.

An alternative (O'Flaherty 1993) is a flow-limited model; this approach quantifies the mass transfer of the extracellular fluid to the tissue compartments of the model. Here, the time variable is incorporated in the flow rates of fluid between body compartments. A central feature of the O'Flaherty model is its emulation of bone growth and resorption as a mechanism for controlling plasma lead levels. "Lead is assumed to instantaneously partition between plasma and soft tissues and to achieve an equilibrium (that is, partition coefficient). Therefore the rates of change of lead masses in soft tissues are limited by the rates of delivery of lead to the tissues, given by the product of the plasma concentration of lead and the rate of plasma flow to the tissue, rather than by limiting steps in the transfer of lead across tissue boundaries" (EPA 2001a).

Predictive blood lead models generally distinguish between the intake of lead during exposure and its uptake by the body. The fraction of lead that is absorbed and enters the blood by whatever portal-of-entry compared with the total amount of lead acquired is termed the bioavailability. In the simple illustration of a PBK model (Figure 6-1), lead intake is represented as ingestion. Subsequently, a fraction of the lead present in the gastrointestinal tract is taken up into the bloodstream—a process that may vary with the age of the individual; the person's health, physiological, and/or nutritional status; and whether ingestion occurred with or without food. Bioavailability of inhaled lead may differ from that of ingested lead. By either route of entry, biokinetic or pharmacokinetic models incorporate a variable for the fraction of total lead that is actually absorbed and define it as the uptake of lead. In the 1999 EPA Guidance Document *IEUBK Model Bioavailability Variable* (EPA 1999), the following terms are defined and adopted for use in this chapter:

- *Absolute bioavailability* is the amount of a substance entering the blood via a particular route of exposure (for example, gastrointestinal) divided by the total amount administered (for example, soil lead ingested).
- *Relative bioavailability* is indexed by measuring the bioavailability of a particular substance relative to the bioavailability of a standardized reference material, such as soluble lead acetate.

Evolution of EPA's IEUBK Model

Federal agencies documented and summarized extensive research on the toxicological impact of lead exposure (McMichael et al. 1986; Bellinger et al. 1989; Bornschein et al. 1989; Needleman et al. 1990; and others)

before development of the IEUBK model (ATSDR 1988; EPA 1989, 1990). As pointed out by Choudhury et al. (1992), epidemiological and behavioral research had not identified a threshold or no-observed-adverse-effect level (NOAEL) that could be used to establish a reference dose for lead—that is, a value that could be used for risk assessment in the manner discussed in Chapter 5 for other metals of concern. Empirical studies showed relationships between children's BLL and the concentration of lead in a variety of media (Barltrop et al. 1975; Yankel et al. 1977; Angle and McIntire 1982; Stark et al. 1982). These slope factor (SF) models were the foundation for the current modeling structure. The impetus for further development of such tools was to quantify the impact of lead in setting National Ambient Air Quality Standards (NAAQS) (EPA 1986) and National Primary Drinking Water Regulations. However, substantial limitations of SF models were identified, owing to the individual variability of children with respect to factors including ingestion rates and activity patterns, the influence of physiological states and nutritional factors on lead absorption, and physicochemical differences in the distribution and occurrence of lead between sites of exposure. Thus, biokinetic models were developed as an alternative approach, emphasizing the need for a predictive capability in order to implement primary prevention strategies.

In 1985, the EPA Office of Air Quality Planning and Standards (OAQPS) began a computer-simulation-model development based on the biokinetic model of Kneip et al. (1983) and Harley and Kneip (1985). These studies brought together a critical mass of biokinetic parameter information. The exposure component for model operation was developed by OAQPS. A 1989 OAQPS staff paper reviewing the NAAQS for lead contained results of model applications to point sources of air lead. Shortly thereafter, the TRW for lead was formed to advise on cleanup at Superfund and Resource Conservation and Recovery Act of 1976 (RCRA) sites; they modified the model for lead risk assessment, calling it the uptake biokinetic (UBK) model. The TRW recognized the desirability of a frequency distribution for BLLs of a population and used a geometric standard deviation based on NHANES II (1986) data.

Initial calibration and validation exercises for the developing model were based on the 1983 Helena, Montana, primary lead smelter study, as cited in the 1989 *Review of the National Ambient Air Quality Standards for Lead: Exposure Analysis Methodology and Validation* (EPA 1989). Further validation of the UBK model was reported by DeRosa et al. (1991) and by Bornschein et al. (1990); whereas the latter study used the Midvale, Utah, data set, the data source for the DeRosa study was not identified. Choudhury et al. (1992) indicated that, for the Midvale exposure data, the UBK default conditions provided an acceptable agreement between observed and calculated values for measures of central tendency but that the upper end of the distribution was not well predicted. Agreement between

predictions and empirical results for Midvale data improved when an age-dependent dust/soil ingestion rate was used. The latter are the same as the current default values for the model. Subsequent to the release of the IEUBK model executable in 1994, additional evaluation of the model was conducted by EPA, including an independent validation and verification of the source code (Zaragoza and Hogan 1998) and an evaluation of predictions of BLLs in children for whom environmental levels and BLLs were measured (Hogan et al. 1998).

The EPA Clean Air Science Advisory Committee (CASAC) of the Science Advisory Board provided initial review and approval of model structure and functioning in 1989. In 1990, CASAC concluded that the model provided "an adequate scientific basis for EPA to retain or revise primary and secondary NAAQS for airborne lead." In 1992, the EPA Science Advisory Board reviewed and reported on the UBK model for lead. Suggested modifications also derived from comments on the draft 1992 Office of Solid Waste and Emergency Response (OSWER) Soil Lead Directive proposed using the UBK model in support of lead exposure risk assessments. Since 1991, the TRW has been responsible for model development. Modifications have made it suitable for evaluating exposure from all media, and the product became a stand-alone PC software package. The biokinetic model approach was deemed suitable for assessing total lead exposures and for developing cleanup levels at residential Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA)/RCRA sites. With refinements resulting from comments on early model versions, the model was released in executable form only in 1994 as the IEUBK model.

DESCRIPTION OF THE IEUBK MODEL

Model Structure and Operation

This section presents an overview of the model's structure and operation. A more detailed summary of the IEUBK model can be found in the work of White et al. (1998). The compartmental structure of the IEUBK model is slightly more complex than that shown previously for the simple PBK example and is illustrated in Figure 6-2 (EPA 1994a). Despite significantly more structure in this version of a multicompartment model, lead accumulation in various model reservoirs still has, as a fundamental control, the time-dependent difference between the uptake and the excretion pathways. When concentrations of lead in environmental media are specified, the model calculates a point estimate of a child's blood lead values over the age range of 0-84 months.

The IEUBK model is defined operationally by EPA's computer program(s). These programs have been publicly available in object code form

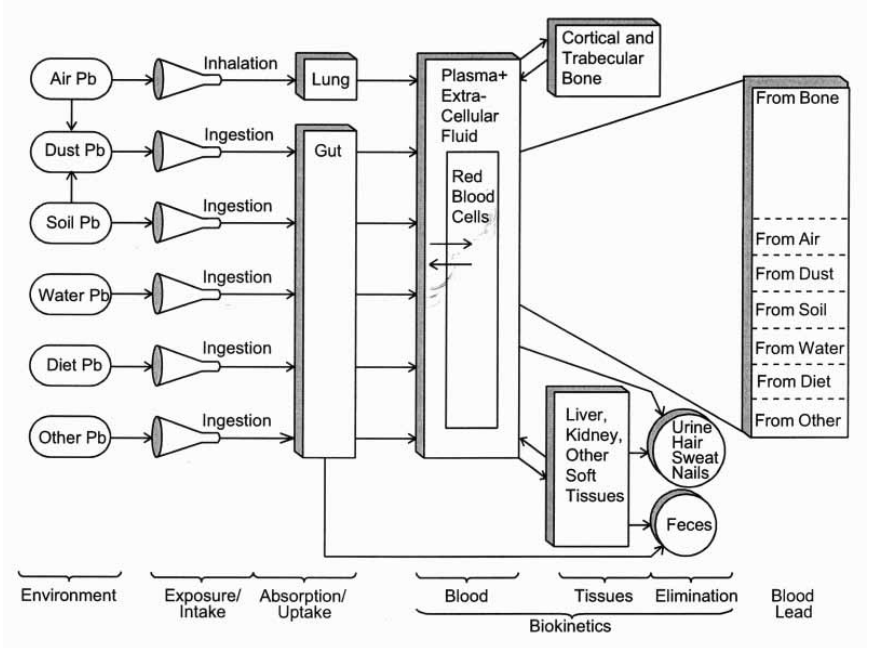

FIGURE 6-2 Compartments and functional arrangement of the IEUBK components for prediction of children's blood lead values. SOURCE: EPA 1994a.

(that is, in a form suitable for running on a computer) since 1994 and have been through multiple versions. The latest version is available from EPA's Superfund Web site (EPA 2004a),[1] and that site also contains technical documentation on the model. The source code for the IEUBK model is not linked at this or any other Web site and has never been readily available in this way; rather, it has always been necessary to specifically request it from EPA.

The primary technical source describing the model is the Technical Support Document (TSD) (EPA 1994b). Although this is explicitly for version 0.99d of the model, the model specification has not changed in any essential way in the 10 years since then. Examination of the computer code shows that the biokinetic portion of the code is identical in all relevant (and some irrelevant) respects. Notably, the current code contains the same

[1]Surprisingly, there appears to be no link to the IEUBK model information from EPA's "lead in paint, dust, and soil" (EPA 2005).

errors[2] and redundancies, as described below, that were present in the original version.

The essential parts of the IEUBK model[3] can be partitioned into four components: an intake component, an uptake component, a biokinetic component, and a probability component. These four components are strictly independent of one another, each feeding into the following one with no feedback.

Intake Component

The intake component of the model collects information on exposures to lead-contaminated media (air, dust, soil, food, water) and sums the quantities of lead that enter the body from each exposure medium. Within each medium, the intake of lead is obtained as the product of an average concentration or mass fraction[4] of lead in the medium and the average intake rate of that medium. For example, the intake of lead from soil is the product of the soil lead concentration (milligrams [mg] of lead per kilogram [kg] of soil) and the ingestion rate for soils (mg of soil ingested per day) to provide an intake rate for lead from soil.

The exposure module contains default values for environmental concentrations and ingestion rates should no site-specific information be available. Similarly, default values for absolute bioavailability are programmed for model operation but may be altered by the user. For soil and dust ingestion, default bioavailability values of 30% are assigned. That value is derived from an absolute bioavailability for soluble lead in water and diet constituents of 50%, together with a 60% relative bioavailability for soil and dust lead compared with water (EPA 1999). Table 6-1 summarizes the IEUBK default values.

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[2]As described in the subsection "Incorrect Model Specifications" below, the committee considers the computer code for the biokinetic part of the model to be in error if it does not solve, in the limit of small time step, the set of algebraic and differential equations and boundary conditions specified in the TSD (EPA 1994b) (which is taken to define the model). The committee has not examined other parts of the code and does not certify that even the examined code is free of other errors. The documentation is considered to be in error if it specifies physical impossibilities or fails to define some element of the model. These definitions are imposed because the committee believes that the model specification should be the standard of comparison (for observations, other implementations, and other models), rather than the computer code itself.

[3]The user interface is not considered here because that does not comprise an essential component of the model. The principal changes in the model over the last 10 years have been in the user interface and in the default values that are automatically present in that user interface.

[4]We do not subsequently distinguish between concentration and mass fraction, using the first term in the usual colloquial sense to represent both.

TABLE 6-1 Default Values for the EPA IEUBK Model

	0-1 y	1-2 y	2-3 y	3-4 y	4-5 y	5-6 y	6-7 y
Ventilation rate, m^3 per day	2	3	5	5	5	7	7
Diet intake, μg lead per day	5.53	5.78	6.49	6.24	6.01	6.34	7.00
Water intake, L per day	0.20	0.50	0.52	0.53	0.55	0.58	0.59
Soil/dust ingestion, total mg per day	85	135	135	135	100	90	85

Water = 4 μg of lead per L, air = 0.1 μg of lead per m^3, maternal blood lead = 2.5 μg of lead per dL.
Indoor air lead concentration = 30% of outdoor concentration.
Soil lead concentration = dust lead concentration = 200 μg lead per gram of soil/dust.
Soil = 45% of total ingestion, dust = 55% of total ingestion.
Diet and water bioavailability = 50%, soil and dust bioavailability = 30%.
NOTE: Bioavailability is not constant. The values cited apply for low lead intake rates. Absolute bioavailability decreases as lead intake increases and uptake saturation is reached.
SOURCE: EPA 1994b.

Uptake Component

The uptake part of the model contains two parts: one deals with absorption in the lung, the other with absorption in the gut. Absorption in the lung is treated as linear; some fixed fraction of the inhaled quantity of lead is assumed to be absorbed. Absorption in the gut is assumed to consist of two fractions: a linear, nonsaturable component and a nonlinear, saturable component. Details of the gastrointestical tract uptake specifications are illustrated in Box 6-2 and Figure 6-3. For each ingested medium (labeled

BOX 6-2 Lead Uptake Formulations for the IEUBK Model

Description of Model Formulation for Uptake of Lead from the Gastrointestinal Tract

Figure 6-3 illustrates the two types of uptake from the gut. Suppose the total lead ingestion intake in medium k is Z_k. Then defining

$$Z = \sum_k \alpha_k Z_k \qquad (0\text{-}1)$$

the linearly absorbed component U_l and nonlinearly absorbed component U_n are assumed to be given by

$$U_t = pZ \qquad (0\text{-}2)$$
$$U_n = (1 - p)Z/(1 + Z/Z_{sat})$$

with the total gut absorption given by the sum $U_l + U_n$. The value p has default value 0.2, and Z_{sat} is estimated by default as 100 μg/day at 24 months, and is scaled with body weight for other ages.

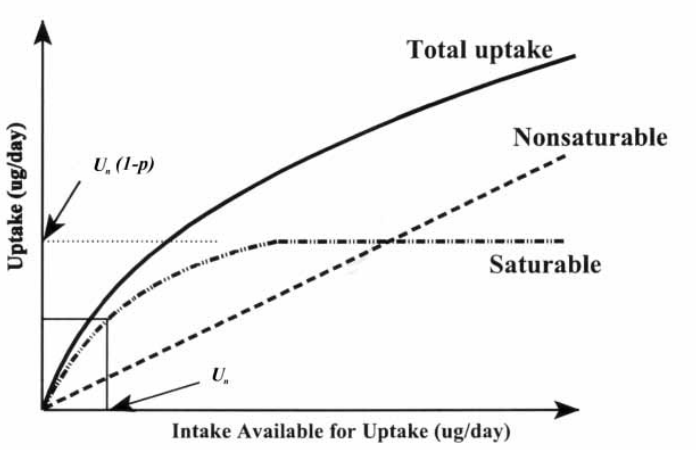

FIGURE 6-3 Mathematical treatment of the lead absorption in the IEUBK model. SOURCE: EPA 1994b.

here by index k), there is assumed to be a fixed fraction α_k (the bioavailability of lead from that medium) that could be absorbed at a low exposure level. The user can override the program default values and specify separate bioavailability values for each exposure medium.

Biokinetic Component

The biokinetic component of the IEUBK model is a compartment model with seven compartments plus three excretion-only pseudocompartments (URINE, FECES, and SNH) as named and numbered in Table 6-2.

The plasma-ECF compartment exchanges lead with all the other compartments, and excretion occurs only to the urine pseudocompartment. The only other connectivity between compartments and pseudocompartments is the excretion of lead from liver to feces and from soft tissues to skin, nails, and hair. The only connection between the uptake and biokinetic components of the model occurs through uptake in the lung and gut. These uptakes are assumed to be independent of the internal state of the body incorporated in the biokinetic component. In theory, there is some dependence—for example because of excretion of lead into the gut (from where it could be re-absorbed) in bile; however, the effect of any such dependencies is expected to be small.

Equations describing the transfer of lead between these compartments (equations of motion) are presented in Box 6-3. Transfer between these compartments is described by the time constants F_i and T_i, which denote uptake to plasma or transfer from plasma, respectively. Similarly, A_i is the

TABLE 6-2 Compartments[a] of the IEUBK Model

Compartment Name	Number	Description
PLECF	0	Plasma-ECF (extracellular fluid)
RBC	1	Erythrocytes
TRAB	2	Trabecular bone
CORT	3	Cortical bone
KIDNEY	4	Kidney
LIVER	5	Liver
SOFT	6	Other soft tissue
URINE	7	Urine
FECES	8	Feces
SNH	9	Skin, nails, and hair

[a]For the compartments, these names are abstracted from the nomenclature used in the documentation and source code of the IEUBK model (EPA 1994b). The compartment numbers are committee constructs. The equations of motion are more compact using this subscript notation.

time constant for the transfer of lead from a compartment to the plasma-ECF compartment or any pseudocompartment. These constants for the different compartments vary with age, and some depend on tissue concentration or are written in such a way as to be related to tissue concentration ratios in order to use experimental data on such ratios. For instance, lead excretion rates vary substantially during a child's early life (O'Flaherty 1993); whereas less than 70% of daily lead uptake may be excreted at age 6 months, more than 90% of daily uptake is excreted at age 24 months. Values of the parameters controlling the transfer processes play a critical role in the accuracy of model predictions. Despite an increase in model complexity (compared with the model structure shown in Figure 6-1), lead accumulation in the IEUBK model compartments is still controlled by the time-dependent difference between uptake and excretion pathways.

The tissue masses (or volumes, for red blood cells, plasma [extracellular fluid], and blood) at each age are defined by mathematical functions that have been chosen to give a good fit to experimental data on tissue masses (or volumes) as a function of age. The masses M_i are supposed to be initialized at age zero to values that give a blood lead concentration of 0.85 times the blood lead concentration of the mother. Equations 0-3 (see Box 6-3) are then integrated over age to obtain the masses of lead in each compartment at any age. Lead concentrations (or mass fractions) in each compartment at each age are obtained by dividing lead mass by tissue volume (or mass) at that age. In particular, blood concentration is obtained by summing the mass in the red blood cells and the mass in the fraction of the plasma-ECF that is in the blood and dividing by blood volume. Finally, the blood concentration value output by the current model user interface is an average over various time periods (for example, the first 6 months of age, 6-12 months, and annual averages to age 7).

BOX 6-3 Equations of Motion for the Transfer of Lead Between IEUBK Model Compartments

The equations of motion for the mass of lead in each of the compartments are as follows:

$$\begin{aligned}
\frac{dM_0}{dt} &= I - \sum_{i=1}^{7} \frac{M_0}{T_i} + \sum_{i=1}^{6} \frac{M_i}{F_i} \\
\frac{dM_i}{dt} &= \frac{M_0}{T_i} - \frac{M_i}{A_i} \qquad 1 \le i \le 6 \\
\frac{dM_7}{dt} &= \frac{M_0}{T_7} \\
\frac{dM_i}{dt} &= \frac{M_{i-3}}{T_i} \qquad i = 8,9
\end{aligned} \tag{0-3}$$

- i compartment number (0-9), from Table 6-2,
- t age,
- I total lead intake rate (mass per unit time) into the plasma-ECF compartment (from the gut and lung),
- M_i for $0 \le i \le 6$ the mass of lead in compartment I; for $7 \le i \le 9$ the cumulative mass of lead excreted to the pseudo-compartment,
- T_i for $1 \le i \le 7$ a time constant for transfer of lead from the plasma-ECF compartment to compartment i,
- T_8 time constant for transfer of lead from the liver to feces,
- T_9 time constant for transfer of lead from soft tissue to skin, nails, and hair,
- Fi for $1 \le i \le 6$, a time constant for transfer of lead from compartment i to the plasma-ECF compartment, and
- A_i for $1 \le i \le 6$, a time constant for transfer of lead from compartment i to the plasma-ECF compartment or any pseudo-compartment.

Only the liver and soft tissue compartments excrete lead (to feces and to skin/hair/nails, respectively; excretion in urine is treated as a transfer from the plasma-ECF compartment), so for compartments 1 through 4 the only exchange is with the plasma-ECF, leading to:

$$A_i = F_i \qquad 1 \le i \le 4, \tag{0-4}$$

and for compartments 5 and 6 it is assumed that

$$1/A_i = 1/F_i + 1/T_{i+3} \qquad 5 \le i \le 6. \tag{0-5}$$

Probabilistic Component

The fourth component of the IEUBK is the probabilistic component. The deterministic estimates of blood concentrations obtained as just described are assumed to represent the median values for a lognormal distribution of values that would occur in a population that was subject to fixed

lead concentrations in the input media (soil, dust, air, water) equal to those input to the model. The standard deviation (or geometric standard deviation [GSD]) of the lognormal distribution was derived based on observations of exposed populations of children. EPA (1994a) stated that the default value of the GSD is based on analyses at Midvale, Utah; Baltimore, Maryland; and Butte, Montana. The analyses are not available for review.

Issues Associated with Using the Model

The statement of task directed the committee to address whether the design, input data, and assumptions of the IEUBK model were consistent with current scientific understanding. Issues associated with IEUBK model predictions of blood lead values can be grouped into three categories: (1) the computer code implementing the mathematics of model computations, (2) the default exposure values related to ingestion rates and to bioavailability of lead, and (3) extension of a deterministic, point value for blood lead concentration to a probability distribution function for a population. Although the model has been subjected to several evaluation and critique efforts, as well as to EPA Science Advisory Board reviews, no comprehensive published account of the peer review content is available. Therefore, a variety of comments on these several categories of uncertainty seem warranted.

Incorrect Model Specifications

With regard to the first category, the TSD has contradictory claims as to the numerical method used to integrate the equations (EPA 1994b). On page 45 of the TSD, the backward Euler scheme is discussed, whereas on page A-14 there is the claim that "These differential equations are translated into difference equations employing the forward Euler solution in the series B-6.5a to B-6.5i, then to the solution algorithm for differential equations using the backward Euler method, or alternate difference equation scheme." It is not clear what this means, or whether any consistent approach was used. The equations given in the TSD agree with a backward Euler scheme except for equations B-8c and B-8d, but the difference for those equations is second order in the time step, the same as the error in any such first-order scheme.

Further, the scheme indicated in the TSD is not actually carried out in the computer program. Rather, it evaluates all age-dependent functions used in the coefficients of the differential equations (in defining the time constants) at monthly intervals and assumes that those values are constant throughout each month. The integration time step (about one-sixth of a day) is then applied to these functions that remain constant for a month at a time. The choice of a first-order integration method must also be questioned, particularly when the time step is left to the user. A better approach

would be to use one of the many standard numerical integrators that allow specification of the allowable error and require the error to be trivially small. Careful review of the model implementation code reveals a number of additional inconsistencies or minor errors in the formulation of the equations documented in the TSD. These are detailed in Appendix C. Combined with the points enumerated above, however, the cumulative uncertainty in computed results is no more than a few percent. Nevertheless, the documentation should accurately reflect the programming.

Uncertainty in Key Default Parameters

Soil/dust ingestion rates and lead bioavailability are two key variables the user may specify in making blood lead value predictions with the IEUBK model. Its default age-specific ingestion rates have remained unchanged since before the 1994 release of the model (Choudhury et al. 1992). Large uncertainties exist in measures of the central tendency for these exposure media ingestion rates by children. Binkowitz and Wartenberg (2001), in their review of literature reports on the subject, showed rates between 10 and 1,000 mg per day for children, with a median value of about 100 mg/day. Little consistency has been shown in the methodological approaches used; variations exist in the media being estimated, the time period used in the observations, and the analytical chemistry techniques of the measurements. Lee and Kissel (1995) suggested a slightly narrower range at a factor of 2 and highlighted the importance of studies to refine ingestion rate values.

Lead bioavailability as a function of age is not well characterized, although there is general agreement among many investigators that bioavailability in pediatric populations is generally higher than it is for adult populations (O'Flaherty 1995; Pounds and Leggett 1998). Although the animal studies of Quarterman and Morrison (1978) supported this view, Mahaffey (1998) urged caution in this interpretation from the limited study data that exist. In the model of O'Flaherty (1993, 1995, 1998), bioavailability is estimated in the 50-60% range for children under the age of 2 years, declining to the 10-20% range by age approximately 5 years. The latter values are similar to those for adults (Maddaloni et al. 1998). The IEUBK default values for soil and dust bioavailability are 30% and are constant across age groupings of children (except see footnote *a* in Table 6-1). Uncertainty in ingestion rate and in bioavailability has a strong, direct influence on the model results.

Uncertainty in Projecting Point Estimates into Population Distributions

One of the more contentious issues associated with the predictive capability of the IEUBK model is the choice of a GSD. The IEUBK model is

designed to predict one BLL for a given set of exposure conditions, and this BLL is designated as the geometric mean of a population of children who would be exposed to the specified environmental levels. The GSD is then used together with the predicted geometric mean to estimate a range of BLLs that might arise in this population. Contention arises in part because EPA's blood lead target of protecting 95% of such a population at a BLL of 10 μg/dL means that the outcome, either in predicted 95th percentile blood lead or in estimated soil lead cleanup level, is very sensitive to the value of the GSD. EPA materials (EPA 2002) state that the GSD should not be site specific because it represents variability in exposure and behavioral parameters outside of soil and dust lead variability and therefore should not change significantly, at least in large populations, from site to site. Although EPA's *IEUBK Guidance Manual* (1994a) specifies a default value for the GSD and states that it is based on calculations at three sites, material documenting these calculations is not in the public domain and therefore cannot be examined or verified.

Although EPA argues strongly for use of the default GSD value, several EPA risk assessments (EPA 1995 [Sandy], 1998a [Palmerton]; Life Systems, Inc. 1995 [Bingham Creek]) have developed and used alternative values of the GSD, leading to the concept that the GSD may be site specific. In the Vasquez Boulevard and Interstate 70 health risk assessment (EPA 2001b), uncertainty in IEUBK model predictions was examined specifically with regard to dietary lead, soil-ingestion rate, and GSD. The report suggested that the default GSD of 1.6 might be too high for this site. Accurate calculation of a site-specific GSD value is a complex procedure (Griffin et al. 1999) involving significantly more effort than a simple analysis of blood lead results; this perhaps underscores EPA's approach to the use of alternative GSD values in IEUBK applications.[5] However, the apparent disparity between stated policy at the federal level and (some) implementations at the regional level can lead to confusion on the part of risk assessors/managers as well as the general public. The economic consequences associated with an inaccurate GSD used for setting cleanup levels can be substantial and a more objective, scientifically comprehensive policy needs to be articulated. A fully probabilistic version of the IEUBK model, such as was demonstrated at EPA's 1999 workshop[6] (see Box 6-4), would estimate the variability in

[5]EPA states, "Model users should not substitute alternate values for the default GSD without detailed site-specific studies designed to document the difference that would justify changing the default value" (EPA 2002).

[6]This version did not incorporate any variability in the biokinetic portion of the model, although it is unclear whether there is any substantial variation in this component at lead intakes corresponding to blood levels of concern at Superfund sites. It is technically straightforward to incorporate such biokinetic variability, although obtaining experimental data for any but the simplest estimates of its size may be infeasible.

BOX 6-4 EPA IEUBK Workshops

EPA has held three workshops focusing on the development and use of the IEUBK model. These workshops include Lead Model Validation (1996), Modeling Lead Exposure and Bioavailability (1998), and Probabilistic Risk Assessment and Biokinetic Modeling (1999). Publications based on presentations at the first workshop are in a supplement to Environmental Health Perspectives (Vol. 106, Supplement 6, December 1998), including a preface by Grant and others stating that the key outcome of the workshop was the establishment of requirements and procedures for model validation.

Although manuscripts were collected from the presenters at the two subsequent workshops in 1998 and 1999, no proceedings have ever been published. The 1998 workshop focused on exposure parameters and produced general consensus among attendees that regulators and industry scientists should work together to reduce uncertainties in the model to improve the accuracy of BLL predictions. Recommendations formed at the workshop included the need to analyze soil and dust samples in multiple ways to better understand bioavailability, the need to develop an improved methodology for differentiating exposure to soil versus dust, and the need to conduct detailed adult soil-ingestion studies.

The 1999 workshop focused on efforts by several groups, including EPA, in developing a fully probabilistic blood lead prediction model. General consensus among attendees was that a fully probabilistic model would aid in understanding how the variability in exposure affects the range of BLLs. EPA presented early work toward developing an "all ages" model. From all appearances, there has been little to no follow up on the work or recommendations regarding the development of a fully probabilistic blood lead prediction model.

BLLs as a function of the variability in all exposure and environmental parameters and would obviate the need for such an ad hoc approach as tacking on a GSD at the end of the calculation in the current version of the model. A fully probabilistic version of the IEUBK model would also end the debate about the extent to which the GSD may be site specific because it could be estimated mathematically for each site.

Model Performance Assessments

Comparison with Other Model Structures

Part of the committee's statement of task was to address whether alternative tools were appropriately used to assess and interpret the model results. The committee found little evidence in the human health risk assessment (HHRA) or in the record of decision (ROD) for the Coeur d'Alene River basin that alternative tools were used to interpret and assess model results. In the absence of this analysis, we examined the Agency for Toxic

Substances and Disease Registry (ATSDR) OU-3 Public Health Assessment (ATSDR 2004, public comment version) and the Heath Consultation (ATSDR 2000a) that did incorporate an analysis of different methodologies.

The ATSDR (2000a) Health Consultation evaluated lead-exposure risks for children living in the Coeur d'Alene River basin (operable unit 3 [OU-3]) based on the environmental lead sampling carried out at residential locations within the basin as targeted by Field Sampling Plan Addendum 6 (FSPA06) conducted in support of the remedial investigation (URS Greiner, Inc. and CH2M Hill 2001). ATSDR used three screening methodologies to predict exposure risk as displayed by blood lead distributions, assuming the exposure environments sampled to be representative of those occupied by children basin-wide. These included the biokinetic SF model of the OMOEE (1994, 1996), the multiple linear regression SF model of ATSDR (1999), and the multicompartment IEUBK model of EPA.

The results from the ATSDR (2000a) comparison of these models indicated that between 22.5% (ATSDR model) and 79% (OMOEE model) of the basin homes sampled have environmental lead concentrations high enough that children in the 1- to 2-year age group would have lead exposures expected to produce BLLs greater than 10 μg/dL. As employed in the ATSDR Health Consultation (2000a), the IEUBK model predicted an intermediate result; 40% of children[7] would be expected to have blood lead exceeding the CDC guideline. In reviewing this study, the committee recognized that the exposure parameters were not standardized between models in this analysis. To address this shortcoming and make further comparisons between these models, additional analyses were conducted on the FSPA06 data set (see Appendix D). First, results using the model input parameters from the original ATSDR (2000a) study were generated. Then, the results were recalculated after input parameters to the different models were standardized to provide similar exposure regimes. Additionally, the models were run using the input parameters from the "box" model used in OU-1 and OU-3 of the Coeur d'Alene River basin. The comparisons were further extended by including predictions from the physiologically based pharmacokinetic model of O'Flaherty (1993, 1995, 1998). These analyses were conducted on 75 homes from the FSPA 06 data set that had both soil and dust lead measures. Details of the methodology comparison are presented in Appendix D.

[7]An important difference in the results from the comparison of models presented in the ATSDR (2000a) study is that IEUBK model output was apparently generated for children 7-84 months of age, not 1- to 2-year-olds as is presented for the ATSDR and OMOEE models. Further comparisons conducted by the committee (presented below) generate output for children of approximately the same age.

Table 6-3 summarizes the results of the model estimates derived from this work. It presents the percentages of children in the 1- to 2-year age group who would exhibit blood lead values below the CDC (1991) level of concern—10 μg/dL—as predicted by the four models using the seventy-five homes' data as residential environments. Its purpose is to compare model results based on realistic environmental lead-exposure potential. Column 1 shows the recalculated results for the 75 homes' data, utilizing the model parameters originally used in the ATSDR Health Consultation (ATSDR 2000a). Column 2 contains results where the OMOEE model ingestion rates were adjusted to match those of the IEUBK default values, recommended ATSDR regression model uncertainties were applied, and IEUBK predictions were targeted for the 12- to 24-month age class. Column 3 entries were computations based on the Bunker Hill Superfund site box model conditions for the IEUBK model detailed above.

The results indicate that the original computations (column 1) were biased by the high ingestion rates applied to the OMOEE model computations. When column 2 results are compared, the range of predictions is substantially reduced. Here, the IEUBK default model predictions are the most conservative (predict the highest BLLs in children).

As noted earlier, SF models, such as the ATSDR and OMOEE models, have significant limitations in their applicability. Multicompartment models in which exposure and biokinetic parameters can be adjusted for site-specific conditions overcome many of these limitations. Very close agreement is achieved for predictions by the two multicompartment biokinetic models (the IEUBK and O'Flaherty model; see Box 6-5). Although this may be expected owing to the common or similar data sets used in model calibrations, the two models used very different computation strategies. The small differences between the IEUBK and the O'Flaherty model results in column 3 are related to the shapes of the bioresponse curves. The O'Flaherty model predicts blood lead for a 2-year-old that is slightly higher than that predicted by the IEUBK model, but it predicts lower values than the IEUBK model for children ages 3-7 years. When averaged by 12-month age classes, the two models agree within less than 5%.

APPLICATION OF IEUBK TO OU-3 (COEUR D'ALENE RIVER BASIN)

Use of the IEUBK Model in a Regulatory Context

The IEUBK model has two uses. The first is to estimate BLLs arising from site-specific environmental lead levels, taking into consideration any relevant site-specific information such as soil lead bioavailability or altered exposure parameters. If those BLLs are found to be elevated above acceptable levels, the second function of the model is to calculate a soil lead

TABLE 6-3 Blood Lead Values for Children in the 1- to 2-Year-Old Group

Model	Column 1 Original ATSDR Health Consultation Input Parameters, Recalculated for 75 RI/FS Homes (% of individuals with BLLs < 10 μg/dL)	Column 2 Adjusted for IEUBK Default Ingestion Rates and 1-2 Year Age Class (GM [GSD] in μg/dL) (% of individuals with BLLs < 10 μg/dL)	Column 3 Same as Column 2, Except Adjusted to BHSS Box Model Conditions (GM [GSD] in μg/dL) (% of individuals with BLLs < 10 μg/dL)
ATSDR[a]	73% < 10.0 μg/dL	9.79 (1.8) 56% < 10.0 μg/dL	8.90 (1.6)[b] 63% < 10.0 μg/dL
OMOEE[c]	20% < 10.0 μg/dL	9.70 (2.0) 53% < 10.0 μg/dL	5.29 (1.8)[b] 89% < 10.0 μg/dL
O'Flaherty		9.84 (1.5)[d] 56% < 10.0 μg/dL	8.40 (1.5)[e] 71% < 10.0 μg/dL
IEUBK	60% < 10.0 μg/dL	11.9 (1.6)[f] 37% < 10.0 μg/dL	7.93 (1.5)[g] 73% < 10.0 μg/dL

Abbreviations: BLLs, blood lead levels; GM, geometric mean; GSD, geometric standard deviation; RI/FS, remedial investigation/feasibility study.

NOTE: Predictions by ATSDR (1999), OMOEE (1994), O'Flaherty (1998), and IEUBK models used paired soil and dust environmental lead data from 75 RI/FS homes (in FSPA06) (see Appendix D). Models included EPA default lead intake values from diet and inhalation (air), and water lead at 4 μg/L except where higher values were measured.

[a]The ATSDR regression model calculates a maximum blood lead value using an uncertainty of the soil and dust SF. In the Health Consultation, the uncertainty was specified as ±1 standard deviation. In columns 2 and 3 of this table, an uncertainty of ±3 standard deviations is used to correspond with the original ATSDR regression model description.

[b]Soil and dust concentrations were set at 60% of the box model values to compensate for reduction in bioavailability to 18%.

[c]The Ontario Ministry of Energy and Environment (OMOEE) model calculates an intake of concern (IOC), not a blood lead value, but this tabulation can be expressed as a percentage of predicted blood lead levels < 10.0 μg/dL. The (estimated) BLLs assumed two times the IOC is equivalent to 10.0 μg/dL.

[d]Soil and dust ingestion rates are fixed program functions; they peak at about 135 mg/day at age 2 but decline subsequently more rapidly than those of the IEUBK model. The integrated soil plus dust ingestion rate is about 65 mg/day over the interval 0-84 months of age.

[e]Model parameters were adjusted to reflect the 60% soil to 40% dust ingestion ratio and the weighted soil concentrations of the box model.

[f]Batch mode IEUBK runs were specified for age 20 months. This produces a blood lead value equivalent to the normal mode blood lead concentration tabulated for the 1-2 year age class.

[g]Results for IEUBK and O'Flaherty models (column 3) do not have statistically different geometric mean values at the 95% confidence level.

BOX 6-5 Multicompartment Biokinetic Models Compared Well

Under the conditions of this comparison, cleanup levels determined by the two multicompartment models would be the same. This supports the veracity of IEUBK biokinetic computations as used in this case. It does not, however, provide a validation of the exposure/bioavailability assumptions used in the operation of these models.

cleanup level that will be adequately protective of young children in the community, such that BLLs will not exceed the established acceptable levels.

Calculation of the soil lead cleanup level requires two items, one mathematical and the other involving policy. The IEUBK model provides the mathematical relationship between environmental lead levels and BLLs that form the basis for the soil lead cleanup level. However, the level of lead in blood that is considered acceptable is equally critical to the calculation of a soil lead cleanup level, and this is a policy decision.

The 5% Criterion

EPA's current policy concerning acceptable BLLs is best articulated in its 1998 Office of Solid Waste and Emergency Response (OSWER) directive (EPA 1998b, see additional discussion in next section). EPA's policy is one of protecting the individual child and states that no child should have greater than a 5% probability of having a BLL above 10 μg/dL. (Note that this target is sometimes referred to as a "probabilistic" target. This is distinct from the IEUBK model itself, which, in its current form, is not probabilistic.) A careful reading of previous OSWER directives (1994 and 1992) and draft directives on this topic suggests that the current policy has always been EPA's policy; however, poor articulation of the statement combined with a lack of understanding on the part of many responsible parties and EPA project managers have led previous applications of the IEUBK model to calculate a soil lead cleanup level consistent with a target of having no more than 5% of the community with BLLs above 10 μg/dL. Indeed, in the Coeur d'Alene River basin, this may be particularly true as the remedial action objective of the cleanup in the box was explicitly stated as 5% of the population.

These targets are sometimes described as "community" and "individual" protection targets, where the community target requires that 95% of children in the community have BLLs below 10 μg/dL, and the individual target requires that each individual child have a 95% probability of having a BLL below 10 μg/dL. Again, although the community protection target

has been adopted at some sites, EPA's policy is to use the individual protection target. One reason to debate the appropriate target of protection is that the choice can have a large impact on the soil lead cleanup level. A community level target will yield a higher soil lead cleanup level for any given site because it is necessary to ensure only that 95% of the community would be expected to have BLLs below 10 μg/dL. Some of these 95% of children with BLLs below 10 μg/dL would be living on yards contaminated just at or below the soil lead cleanup level, whereas (many) others would be living on yards with lower soil lead levels. The individual protection target is stricter than the community protection target in that it requires that 95% of children who live where they are exposed to maximum levels of lead in soil (at the soil lead cleanup level) will have BLLs below 10 μg/dL. The entire 95% of children with BLLs below 10 μg/dL would be equally exposed to yards contaminated just at or below the soil lead cleanup level. Again, this distinction is one of policy, and neither target is scientifically correct or incorrect.

Application of the Geometric Standard Deviation

One of the most critical parameters required in calculating the soil lead cleanup level is the individual blood lead GSD. The individual GSD expresses the range of BLLs that can arise due to all factors other than a narrow range of environmental lead concentrations.[8] These factors include behavioral components, such as soil ingestion rates, biokinetic differences between individuals, and ranges of lead intake from sources other than the site, such as food. The value of the individual GSD is necessarily less than the value of a community GSD, derived from the range of BLLs seen in a community. The community GSD must be higher because, in addition to all the components that contribute to the individual GSD, the community GSD also includes a component of variability due to variable environmental concentrations. The IEUBK includes a recommended default individual GSD,[9] although site-specific blood lead data have been used at some sites to alter its value (EPA 1995, 1998a; Life Systems 1995). The individual GSD is also used to estimate the percent of BLLs greater than 10 μg/dL in an

[8]EPA states that the GSD is not intended to address variability "in blood lead concentrations where different individuals are exposed to substantially different media concentrations of lead" (EPA 1994a).

[9]A fully probabilistic version of the IEUBK model, such as the ISE model, would calculate a site-specific individual GSD a priori. Such a probabilistic approach would reduce uncertainty associated with the default recommendation for the GSD and would obviate the need for large amounts of site-specific blood lead data to calculate a site-specific GSD using the current model approach.

IEUBK model prediction. If this percent agrees well with observation (considering all the limitations of such comparisons discussed below), then this is an indication that the GSD value may be appropriate for the community.

Once an adequately predictive model of the relationship between environmental lead and blood lead in a community has been developed, including the GSD, and the target level of protection has been chosen, the IEUBK model can be used to calculate the soil lead cleanup level. This is done as follows: if we assume that no individual child should have more than a 5% probability of a BLL exceeding 10 μg/dL, and we use the individual GSD model-recommended value of 1.6, we can then calculate that this requires a geometric mean (GM) BLL of 4.62 μg/dL from the following relationship:

$$10\ \mu g/dL = GM \times \exp(1.645 \times \ln([GSD]).$$

The IEUBK model is then run to find the soil lead concentration that yields a predicted geometric mean blood lead of 4.62 μg/dL. Note the overall conservativeness of this approach—EPA's target requires a predicted geometric mean BLL of 4.6 μg/dL for children living on the highest soil lead concentration left unremediated. This is the reason that communities are sometimes identified for lead remediation when no children have BLLs above 10 μg/dL. This level of protection stems from policy decisions; as such, they are not under the purview of this committee considering scientific and technical aspects.

Interpretation of the OSWER Directives

EPA issued an OSWER directive in 1998 (EPA 1998b) that specifies use of the IEUBK model for lead risk assessment for young children and describes EPA's policy concerning acceptable BLLs and the relationship of modeling to blood lead studies. This OSWER directive is an update of an earlier directive issued in 1994.

The 1998 OSWER directive articulates EPA's policy of protecting an individual child from having more than a 5% probability of a BLL elevated above 10 μg/dL (see discussion above). The 1998 OSWER directive also makes clear that EPA views blood lead data alone as insufficient for performing a risk assessment, stating "that predictive tools should be used to evaluate the risk of lead exposure, and that cleanup actions should be designed to address both current and potential future risk." The insufficiency of blood lead observations alone is linked to the policy of protecting individual children, because blood lead information without accompanying environmental lead levels cannot adequately assess the exposure potential that exists, and information about today's blood lead concentrations is insufficient to address what BLLs might occur for other current and future

children exposed to the same environmental lead concentrations. The 1998 OSWER directive stresses the interpretive utility of comprehensive blood lead studies, which include an exposure assessment component, over simple blood lead screening or monitoring program observations (see discussion in Chapter 5). Nevertheless, "OSWER recommends that blood-lead studies not be used to determine future long-term risk where exposure conditions are expected to change over time."

Unfortunately, the OSWER directive's stated preference for IEUBK-calculated BLLs over actual observation for risk assessment purposes has been misinterpreted by the public, which does not always understand the need for risk assessment or remediation in the face of community BLLs that do not appear to be substantially elevated, and by some EPA project managers who, as a result, ignore or downgrade the importance of valid blood lead information. There is almost never a situation in which model predictions are more accurate than a representative set of observations. EPA should clarify that the IEUBK model is preferred because it does two things that blood lead information alone does not do: it mathematically describes the relationship between environmental lead levels and BLLs, and, because of that description, it allows the calculation of a soil lead cleanup level that will be sufficiently protective.

It should also be made more clear that blood lead observations can be very useful and should not be discarded during the risk assessment process. The OSWER directive acknowledges this with the following:

> Blood-lead data and IEUBK model predictions are expected to show a general concordance for most sites. However, some deviations between measured and predicted levels are expected. On some occasions, declines in blood-lead levels have been observed in association with lead-exposure reduction and health education. However, long-term cleanup goals should be protective in the absence of changes in community behavior as there is little evidence of the sustained effectiveness of these education/intervention programs over long periods of time. …Where actual blood-lead data varies significantly from the IEUBK Model predictions, the model parameters should not automatically be changed. In such a case, the issue should be raised to the TRW to further identify the source of those differences.

However, little guidance is available about what to do if IEUBK model predictions and blood lead data do not match other than to consult the TRW. It is clear that the blood lead observations should not be ignored in such a case, provided a representative sample of children has been surveyed. It is particularly important that a protocol for comparison between observed and predicted results should be standardized for risk assessment purposes to prevent further confusion being added to the interpretive pro-

cess. Hogan et al. (1998) presented two types of comparisons that appear useful.

Development of Risk-Based Exposure Media Concentrations

In the statement of task, the committee is asked to examine whether the model has been appropriately applied given the local and regional characteristics of the Coeur d'Alene River basin. The committee has undertaken an analysis of environmental lead measurements specifically to determine whether EPA's work has been adequate in this regard.

Lead in Soil and Dust

The IEUBK model calculates the intake of lead derived from the incidental ingestion of contaminated outdoor soil and indoor dust as the weighted intakes of the respective soil/dust particles and the concentrations of lead in those exposure media. Although this formulation is straightforward, the underlying processes controlling children's exposures to environmental lead are complex. One of the primary links in the transfer of lead in soil and dust to the gastrointestinal tract is the hand-to-mouth behavior of children. Some of the soil and dust that hands come in contact with ends up adhering to them, and subsequent activity transfers hand-adhering dust to the mouth. Two important properties of lead-bearing dust and soil must be addressed to determine the appropriate concentrations for use in the IEUBK model and the associated sampling protocols. The first is the particle-size dependence of concentration of lead in surficial dusts and soils and the other is the contribution of outdoor soil lead to indoor lead in household dust. Both of these influence the parameter values used in the IEUBK model applications to represent the source of the exposure. Model default values appear to show percentages of time that a child is in contact with soil or dust, but, in fact, they simply establish an exposure weighting for these two sources.

Investigators have shown that fine particles, especially those less than 100 micrometeres (mm) in diameter adhere more strongly to hands (Duggan et al. 1985; Duggan and Inskip 1985; Sheppard and Evenden 1994; Kissel et al. 1996) and that, as particle size increases, adherence to skin decreases. According to EPA (2000), the upper bound of the size fraction adhering to skin is 250 μm, based on a review of several studies dealing with dermal contact with soil. The so-called "fine" fraction of a dust and soil sample (defined as particles less than 250 μm) is also likely to be enriched in lead compared with lead in the bulk soil sample. EPA's guidance for the sampling and analysis of lead-contaminated soils recommends that the maximum sieve size for such soil is 250 μm (a No. 60 sieve) (EPA 2000).

However, the guidance also states that other sieve sizes may be used but warns that lead enrichment is likely to increase with smaller sieve sizes. Soil and dust sampling programs in the Coeur d'Alene River basin that are the source of the data used in IEUBK model runs, in contrast, have relied on a standard 175 μm sieve size (a No. 80 sieve). The rationale for this particular sieve size includes compatibility with earlier soil sampling protocols in the Coeur d'Alene River basin and consistency with soil adherence data for dermal exposures (see EPA [2001a] for additional discussion). Although enhanced lead enrichment would be expected in soils processed with the 175 μm sieve instead of the 250 μm sieve, the real issue from a human exposure assessment standpoint is not lead enrichment, but rather the accurate characterization of lead in the particles that play the dominant role in the soil/dust-to-hand-to-mouth pathway. In fact, Gulson et al. (1995) contended that a 100 μm cut point would be preferable for determining concentrations of lead in both soils and dusts.

The transport potential of lead-contaminated soils to the indoor environment by foot traffic and pets is also a function of the size distribution of soil particles and the associated concentration of lead in the various fractions. Specifically, footwear normally would be expected to carry fine-mode particles indoors except under wet conditions; consequently, concentrations of lead and other metals associated with this fraction would be the most closely related to the indoor levels. Once soil-derived particles are tracked into the house environment, a variety of redistribution and dilution processes occur that collectively produce indoor dust. For example, the tracked-in soil mixes with a variety of organic-rich indoor sources such as lint, exfoliated skin, carpet fibers, and dried food particles. Concentrations of organic matter in house dust can exceed 30% by weight (see Fergusson and Kim 1991; Molhave et al. 2000). Consequently, the concentrations of outdoor-derived soil contaminants are lower in indoor dust, provided that there are no indoor sources of the soil contaminants. Dust is distributed throughout a house by foot traffic and by the resuspension of floor particles into household air by walking and by particulate emissions from vacuuming. The airborne particles are then deposited onto floor and nonfloor surfaces and exhausted to outdoor air via normal air exchange processes that also transport outdoor air particles through the building shell into the indoor environment (Schneider et al. 1999).

Epidemiological studies investigating the relationships between blood lead and environmental/socioeconomic parameters have shown that children's contact with lead-bearing household dust (represented by lead loading on floor surfaces, rather than by lead concentration in the dust) is a key determinant of BLLs (see Lanphear et al. 1998). Studies of data specific to the Coeur d'Alene River basin involving blood lead and environmental lead measurements have also supported the important role that indoor dust

plays as an exposure medium for blood lead. Note, however, that the significance of the indoor dust in this context is related to the location of the exposure. Many young children spend more time indoors than outdoors, and outdoor soils may be a major source of indoor lead because of transport of soil particles on footwear and by pets. The Idaho Department of Health and Welfare (IDHW) environmental health assessment conducted for ATSDR (ATSDR 2000b) found that the logarithm of the lead loading rate (in $mg/m^2/day$) on entryway sampling mats explained 46% of the variance in log-transformed blood lead concentrations in children 9 years old and younger. Although this analysis did not control for such confounding factors as lead paint, the results are similar to those of the HHRA (TerraGraphics et al. 2001), which found that lead loading per unit mat area per day was the most important variable in determining blood lead in multiparameter regressions (other parameters included children's age, yard soil lead, and lead paint metrics). Statistical analyses presented in the HHRA (TerraGraphics et al. 2001, Table 6-20) of the relationship between the concentrations of lead in mat dust and other environmental lead measurements indicated that 42% of the variation in mat lead was due to yard soil lead. Other contributors were lead in community soils and interior paint condition.

An important finding of the IDHW environmental health assessment (ATSDR 2000b, Table 4) was that the average lead concentration in mat dust (n = 400, 1,416 μg/g) was nearly a factor of two greater than the average concentration of lead in the yard soils of the houses studied (n = 815, 738 μg/g). Sampling data for entryway sampling mats in houses and yard soils in Coeur d'Alene River basin communities (TerraGraphics et al. 2001, Table 6-11a-j) showed similar results—that is, significant enrichment in mat dust lead compared with lead in yard soils. If outdoor soil is the principal source of lead in indoor dust (and the key environmental medium targeted for remediation), then why is the concentration of lead in entryway dust (as sampled by mats that intercept soils tracked in by residents) significantly higher than that in the outdoor soils?

The answer to this question could result in a better quantitative characterization of the relationship between the concentrations of lead in soil and dusts and associated exposure simulations in the IEUBK model. The committee has analyzed environmental lead, iron, and manganese measurements available from one of the remedial investigation/feasibility study (RI/FS) data sets to further explore the significance of this question, as detailed in Appendix E. Key findings summarizing the significance of additional analyses for source apportionment are as follows:

- Particle size fractionation processes are the most-likely explanation for the average differences in lead observed for soils, entryway mats, and

vacuum cleaner dusts. This emphasizes the significance of evaluating lead concentrations across different size fractions of environmental media in the lead exposure assessment, measurements that EPA did not undertake. Future studies should also address the possibility that perimeter soils containing paint-derived lead represent an additional source of lead in indoor dusts.

- However, the foresight to carry out the bulk analysis for the crustal elements iron and manganese made possible additional evaluations in support of exposure assessment, demonstrating their value for inclusion in the RI/FS investigations.
- The results underscore the significance of soils in the exposure pathway by virtue of their major contribution to indoor dust, providing support for the site-specific exposure parameters used in the IEUBK model runs.

Air Monitoring Data

Exposures to airborne lead can occur by the inhalation of particulate lead in indoor and outdoor air as well as indirectly by hand-to-mouth contact with lead on indoor surfaces that is derived from the deposition of airborne lead that has penetrated the building shell. In general, the inhalation exposure pathway for environmental lead plays a minor role compared with the ingestion of lead in soils and dusts. The IEUBK model includes two default methods for relating concentrations of lead in outdoor air to related levels of lead in indoor air and household dust. The first default is an outdoor level of lead in ambient air of 0.10 $\mu g/m^3$ and an indoor conversion factor of 30% (the indoor air concentration of lead is 30% of the outdoor level). The second default uses a fixed ratio of the concentration of lead in dust to the concentration of lead in outdoor air of 100 μg of lead/g of dust per μg of lead/m^3 in outdoor air. The second default option was not used in the HHRA simulations because direct measurements of lead in residential dusts were used as inputs. In the HHRA, the default value of 0.1 $\mu g/m^3$ air lead concentration was used. Although this value is greater than the expected air concentrations in the basin, the overall contribution of this pathway to absorbed blood leads is just a few percent of the lead intake (EPA 2001b).

Nevertheless, failure to determine the magnitude of airborne inputs to residences can potentially distort the relative importance of alternative transport pathways for the migration of soil-derived lead to the indoor environment and potential sources of variability in BLLs.

As a means of investigating the nature and magnitude of exposures to airborne lead in the Coeur d'Alene River basin, we reviewed historic data on measurements of airborne lead from a monitoring station in Kellogg. Air monitoring for lead started in 1982 and continued until mid-2002 when the

station was shut down. Since smelter emissions ended in 1981, ambient levels of lead have steadily declined (Figure 6-4). The concentration of lead in airborne particles is determined by collecting total suspended particulates on a filter and then analyzing the lead content of the collected particles. The product of the total suspended particulate (TSP) concentration (g/m^3) and the lead concentration in collected particles ($\mu g/g$) gives the ambient lead concentration in air in units of $\mu g/m^3$. So, with data on both TSP and ambient lead levels (the reportable air quality measurements), it is then possible to determine the concentration of lead in suspended particles. Figure 6-5 presents the TSP levels and associated concentrations of lead in ambient particles for the years 1982 to 2001. The most significant features of the graph are the dramatic decline in the lead concentrations in suspended particulate matter and the gradual reduction in TSP.

It is important to point out that, after the end of smelter emissions, the principal source of ambient lead in the atmosphere would be the resus-

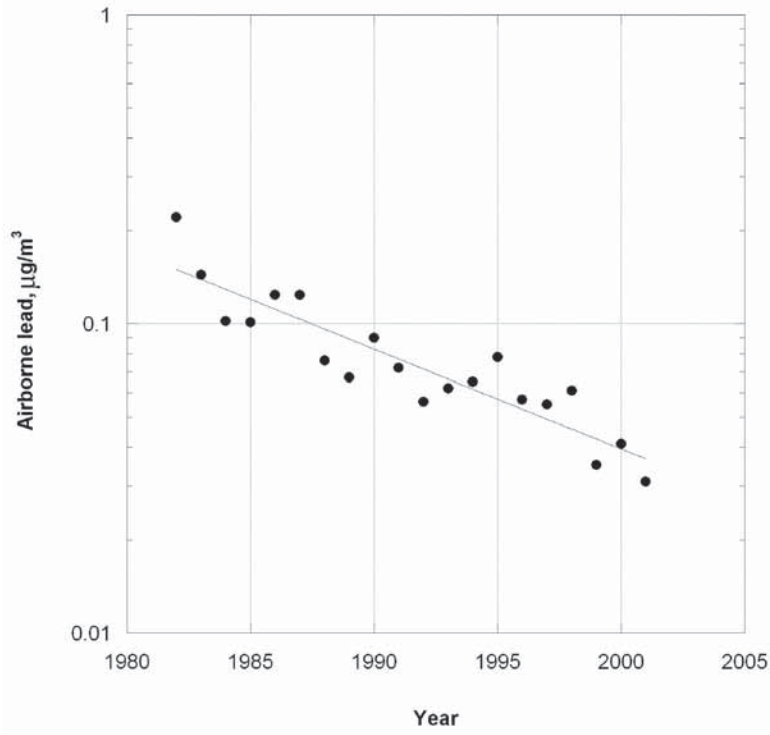

FIGURE 6-4 Concentrations of airborne lead measured at a monitoring station in Kellogg, Idaho, during the years 1982 to 2001. Monitoring ceased in 2002. SOURCE: Idaho Department of Environmental Quality, unpublished material, 2004.

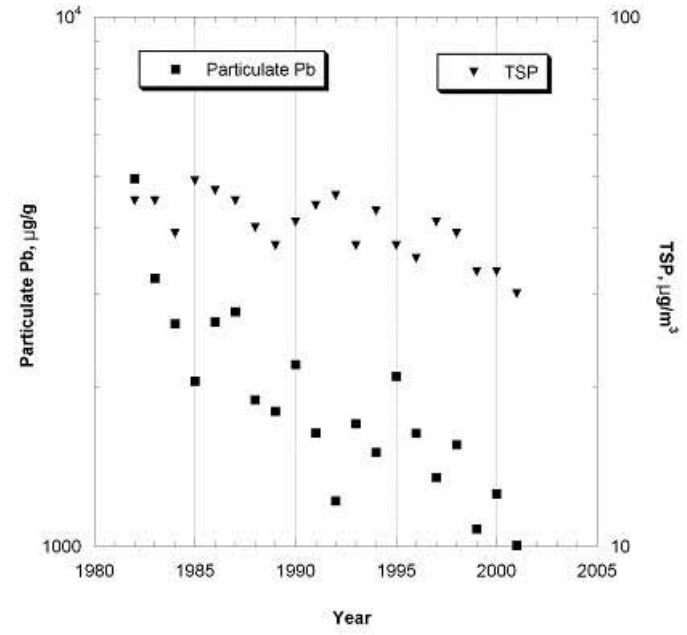

FIGURE 6-5 Long-term trends in the concentrations of lead in suspended airborne particles and mass loading of particles in air. Data are for an air monitoring station in Kellogg, Idaho. SOURCE: Idaho Department of Environmental Quality, unpublished material, 2004.

pension of lead previously deposited from the atmosphere along with wind-driven emissions of dust from surficial soils containing lead derived from previous mining operations. The decline in the concentrations of particulate lead at the Kellogg monitoring site is probably a function of both soil remediation efforts and natural soil weathering processes. But, according to von Lindern et al. (2003a) the major yard remediation work did not begin within the box until 1998; consequently, the substantial declines observed in particulate lead levels before that time depicted in Figures 6-4 and 6-5 undoubtedly are associated with weathering of soil lead.

The phenomena of contaminant weathering of surficial soil contaminants and related declines in airborne loadings has been of particular interest to researchers studying the transport and fate of radionuclides deposited onto the land surface (see Anspaugh et al. 2002). One simple approach for estimating the concentration of a soil contaminant in ambient air is to multiply the TSP level times the concentration of the contaminant in soil and an enhancement factor, which is defined as the ratio of the concentration of the contaminant in airborne particles to the concentration in soil. A

recommended default value for the enhancement factor is 0.7 for soils that are weathered (NCRP 1999). On the basis of this resuspension model, the levels of lead in suspended particles at the Kellogg monitoring site are exhibiting substantial enrichment. In 1995, for example, the community-wide concentration of lead in Kellogg soils was about 1,000 μg/g (von Lindern et al. 2003a), but the airborne particles contained lead at about 2,094 μg/g (see Figure 6-5) or about a factor of 2 higher. Moreover, even though the cleanup goal for yards of 350 μg/g was achieved for residences in Kellogg by 1998, the lead concentration in soil-derived suspended particles for 2001 (about 1,000 μg/g) was nearly a factor of 3 greater! The elevated concentration of lead in airborne particulate matter compared to the levels of lead in bulk soils processed with a 175 μm sieve size provides additional evidence that lead may be preferentially concentrated on fine soil particles—due to previous atmospheric inputs as well as other geochemical weathering processes of mining wastes mixed with Coeur d'Alene River basin soils.

Lead in Drinking Water

The default concentration for lead in drinking water used in the IEUBK model is 4 μg/L; for comparison, the national drinking water action level for lead is 15 μg/L (EPA 2004b). Measured values of lead in drinking water for Coeur d'Alene River basin communities are given in HHRA Tables 6-11a-j (TerraGraphics et al. 2001). Most of the reported concentrations for lead in "first draw" water from taps and private well waters were between 2 and 4 μg/L, although some of the maximum values reported exceeded the action level for lead in drinking water. Concentrations of lead in "purged" samples of tap water were substantially lower than the first-draw samples. For example, in Wallace, the geometric mean concentration of lead in purged water was 0.65 μg/L, compared with 3.19 μg/L for the first-draw samples. Although IEUBK guidance recommends averaging the lead concentrations in the first-draw and purged samples, the HHRA used only the purged values for the batch-mode runs of the IEUBK model. No rationale was given for that decision; however, the consequences are expected to be minor given the relatively small contribution that drinking water provides to overall lead intake. In another example of potential bias, the HHRA notes that levels of lead in well waters are overestimated because the original water analyses taken in 1996 did not report concentrations below the then-current lead drinking water source standard of 5 μg/L. In fact, 183 of 222 wells sampled in 1996 had censored results—that is, values at or below 5 μg/L. Later studies indicated that the geometric mean value for well waters is 0.75 μg/L (TerraGraphics et al. 2001). So, use of the existing concentration values for lead in well waters for the batch mode

IEUBK model would have overestimated drinking water exposures to lead. But again, the consequences are not likely to be significant because of the minor role this pathway plays in the overall intake of lead.

Lead in Local Food Supplies

Dietary intakes of lead were simulated in the HHRA using baseline and incremental exposure scenarios. In the baseline scenario, children consume lead derived from a typical "market basket" of foods, and therefore the default input parameters for dietary lead were adopted. However the default dietary lead intakes in the IEUBK model are based on older data and are higher than would be suggested by more-recent dietary information (Bolger et al. 1996). Therefore, dietary exposure to lead is probably overestimated in the baseline scenario. To estimate dietary intakes for the incremental exposure scenario—designed to represent exposures associated with a limited subset of the population—information is required on both the concentrations of lead in selected foods and related intakes. Residual lead in Coeur d'Alene River basin soils and surface waters can produce elevated dietary exposures to lead for children in households that rely on homegrown produce or locally caught fish for a portion of their regular diets.

Based on sampling conducted as part of the HHRA, the median concentration of lead in fish was 0.12 μg/g wet weight, and the 95th percentile concentration was 0.68 μg/g wet weight. With a fish fillet intake rate of 5.4 g/day (TerraGraphics et al. 2001, Table 6-39), the respective lead intakes for the central tendency (CT) and reasonable maximum exposure (RME) intakes for children were 1 and 4 μg/day. These intakes represent a small increment above the baseline lead intakes that range from 30 μg/day for the lower basin to 99 μg/day for Wallace.

The median concentration of twenty-four samples of homegrown vegetables collected from Coeur d'Alene River basin communities was 3.2 μg/g wet weight (TerraGraphics et al. 2001, Table 6-40a); with an intake of 7.4 g/day of garden vegetables (based on a 15 kg child; TerraGraphics et al. 2001, Table 6-39), the associated CT lead intake is 24 μg/day. At the 95th percentile concentration in garden vegetables (24 μg/g wet weight), the lead intake becomes 178 μg/d (representing the RME estimate). In contrast, the default dietary intake for children ages 1-5 years is approximately 6 μg/day. Although the levels of lead in homegrown produce vary according to the levels of lead in soil, the HHRA uses the same median and 95th percentile intakes for all communities in the incremental exposures used in the IEUBK model (TerraGraphics et al. 2001, Figures 6-21a-h).

The estimated lead intake for the CT exposure case seems plausible; however, the RME intake is not entirely consistent with blood lead measurements. According to Table 6-55b of HHRA, the geometric means of the

BLLs predicted by the IEUBK model for the RME case would exceed 20 μg/dL for both the EPA default and box model implementations. But in Table 6-2 of the HHRA, there were only 12 instances in which BLLs exceeded 20 μg/dL out of 524 measurements made during the years 1996-1999 (about 2% exceedance). It is not possible to determine whether the consumption of homegrown produce was a contributing factor to those exceedences, because household-specific information on dietary practices was not reported. Nevertheless, the available information from several studies suggests that the consumption of homegrown vegetables is unlikely to play a dominant role in causing elevated BLLs. For example, uptake ratios for arsenic and lead into vegetables have been found to be low (Glass and SAIC 1992; EPA/SRC 2001), and biomonitoring data from many sites including the Basin (ATSDR 2000b) have not indicated that ingestion of homegrown vegetables contributes to elevated lead and arsenic exposure in residents (Polissar 1987; Polissar et al. 1990; Bornschein et al. 1991; ATSDR/CDOH 1992; BSBDH and University of Cincinnati 1992; Hwang et al. 1997).

Configuration and Use of the Bunker Hill Superfund Site Box Model

The HHRA utilizes the IEUBK model in four modes. Assumptions are either "default" or "box" and operation is either "community" or "batch." The regression analyses for examining the relationships between environmental lead and blood lead values (TerraGraphics et al. 2001; von Lindern et al. 2003a) provided a basis for the structural equation modeling (SEM) source apportionment. These results indicated that, for the Coeur d'Alene River basin, site-specific deviations from the IEUBK default proportions of soil and dust ingestion should be used. Soil was shown to be the major contributor to the combined exposure medium and should be weighted more heavily than the nonsoil lead contained in house dust. A 60% soil and 40% dust division is supported by the soil tracer element analysis described in Appendix E. The SEM also highlighted the apparent role of community-wide soil lead concentrations in the exposure dynamic. A reduction in lead uptake was indicated by the SEM analysis, and the box model implemented this by reducing the bioavailability values used by the model; default soil/dust ingestion rates were maintained. These adjustments from IEUBK default configurations provided a better fit, for the several possibilities considered, between observed and predicted blood lead values and are contrasted in Table 6-4.

When interpreting the fractions of soil/dust ingestion summarized below, the proportions reflect the source of the materials to which the child is ultimately exposed and not the proportion of time that a child spends in each of these environments. The IEUBK model does not separate the soil and dust ingestion regime with respect to time spent indoors or outdoors. It

TABLE 6-4 Default and Box Assumptions Used in the HHRA

Model	Fraction (%) of Soil/Dust Lead Ingestion Attributed to			Bioavailability of Lead in Soil (%)
	House Dust	Yard Soil	Neighborhood Soil	
Default	55	45	0	30
Box	40	30	30	18

SOURCE: TerraGraphics et al. 2001.

models the combined exposure dynamic using the concentration of lead in the two media and the fraction each contributes, either directly or indirectly, to the daily lead ingestion intake. Soil is very clearly an important constituent of household dust. Details of soil and dust transport as well as children's activity patterns will vary greatly among locations considered, and these inputs to the IEUBK model represent the average way in which the exposure parameters affect the model predictions.

Application of the IEUBK model in batch mode permits limiting simulations to those households for which both environmental and matched blood lead data are available. Evaluation of batch mode IEUBK results, therefore, avoids questions about the representative nature of the overall basin blood lead data set. Batch mode IEUBK predictions (both "default" and "box" versions) and corresponding observations are presented in Figure 6-6a (percent of blood lead ≥ 10 μg/dL) and Figure 6-6b (geometric mean blood lead in μg/dL) for each of the eight study areas. Study areas are placed on the x-axis for these figures in roughly geographical order running from west to east in the basin. Model results both underpredict and overpredict observed values depending on model version and study area.

To facilitate interpretation of data in Figure 6-6a, absolute differences between the predicted and observed sample fraction (expressed as percent) exceeding 10 μg/dL for the default model and the box model are presented in Figure 6-7a and 6-7b, respectively. Bars falling below the x-axis in Figures 3 and 4 reflect underprediction by the IEUBK model and bars falling above the x-axis reflect overprediction. In Figure 6-7a, it can be seen that the default model overpredicts in six of eight study areas and underpredicts in two. In all cases, the magnitude of deviance is greater than 5% of the observations. In contrast, Figure 6-7b shows that the box model tends to better predict the fraction exceeding 10 μg/dL in those areas in which the default model overpredicts but produces greater underprediction in the two most westerly (downstream) study areas.

Examination of differences between predicted and observed geometric mean BLLs as shown in Figure 6-8a and 6-8b reveals a very similar pattern. The default model overpredicts in the upper basin and underpredicts,

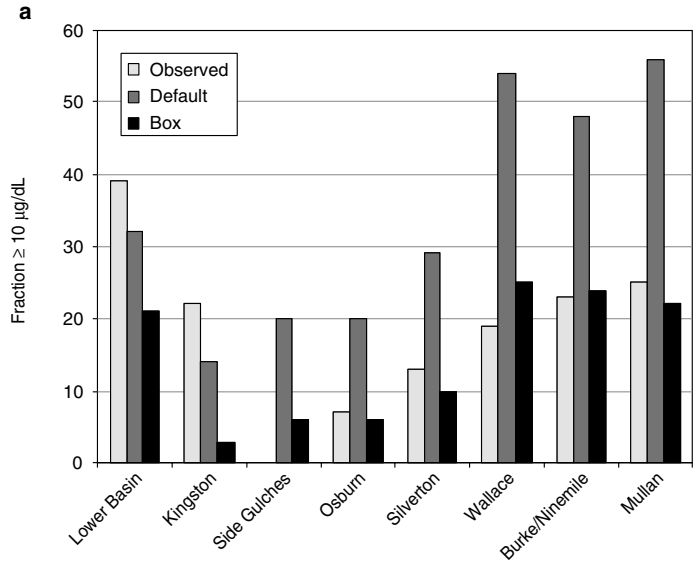

FIGURE 6-6a Fraction exceeding 10 µg/dL by study area for children 1-5 as observed and predicted using IEUBK default and box models in batch mode.

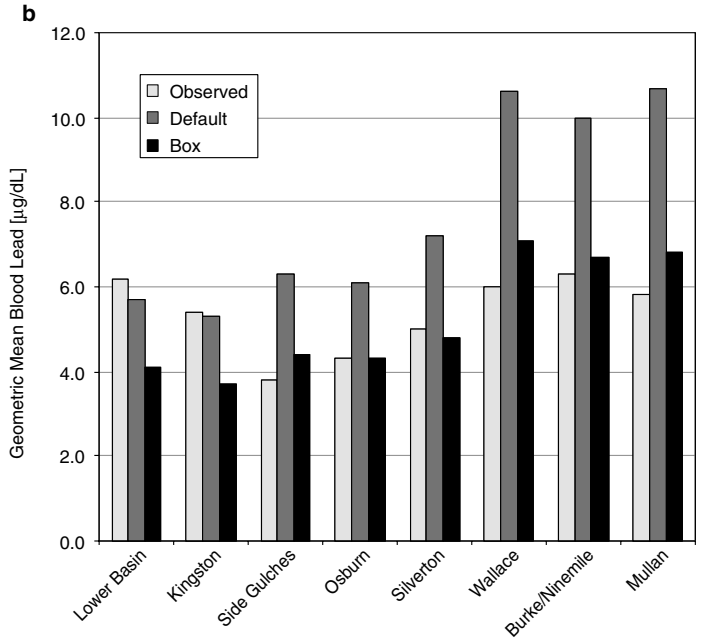

FIGURE 6-6b Geometric mean blood lead (µg/dL) by study area for children aged 1-5 as observed and predicted using IEUBK default and box models in batch mode.

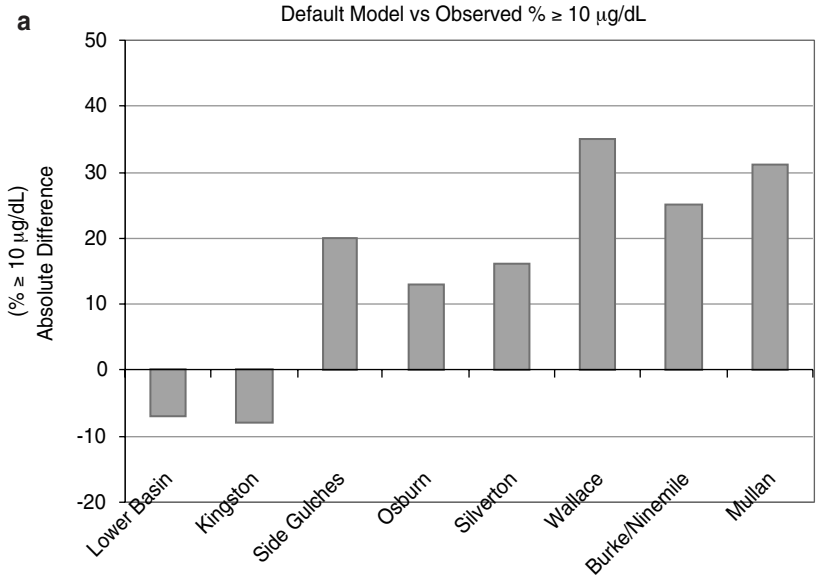

FIGURE 6-7a Absolute differences between batch mode IEUBK default model prediction and observed fraction exceeding 10 μg/dL by study area for children 1-5.

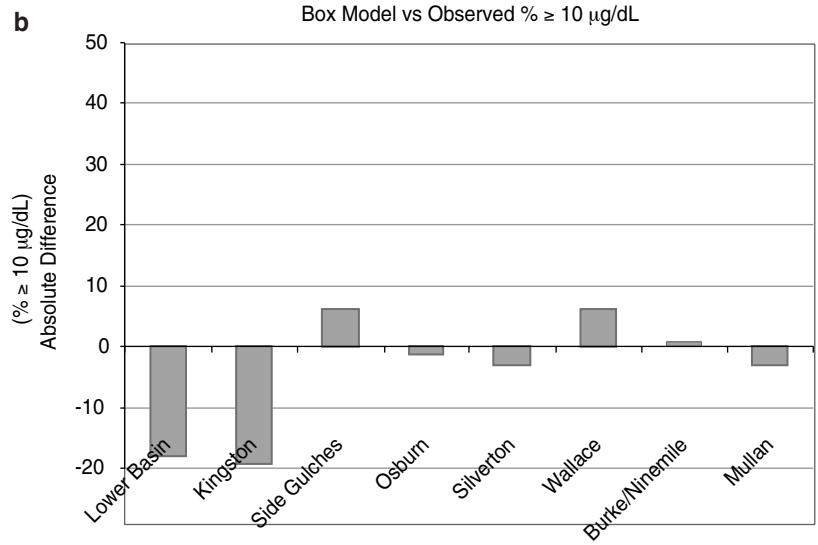

FIGURE 6-7b Absolute differences between batch mode IEUBK box model prediction and observed fraction exceeding 10 μg/dL by study area for children aged 1-5.

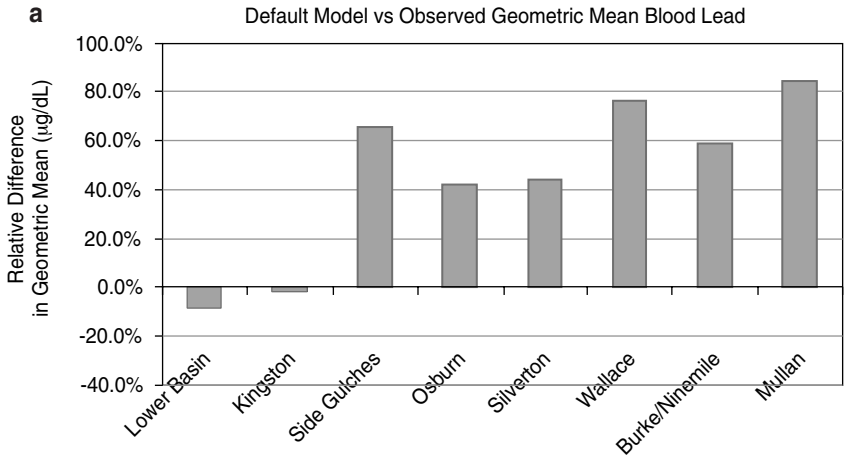

FIGURE 6-8a Relative difference (as percent) between batch mode IEUBK default model prediction and observed geometric mean blood lead (µg/dL) by study area for children 1-5.

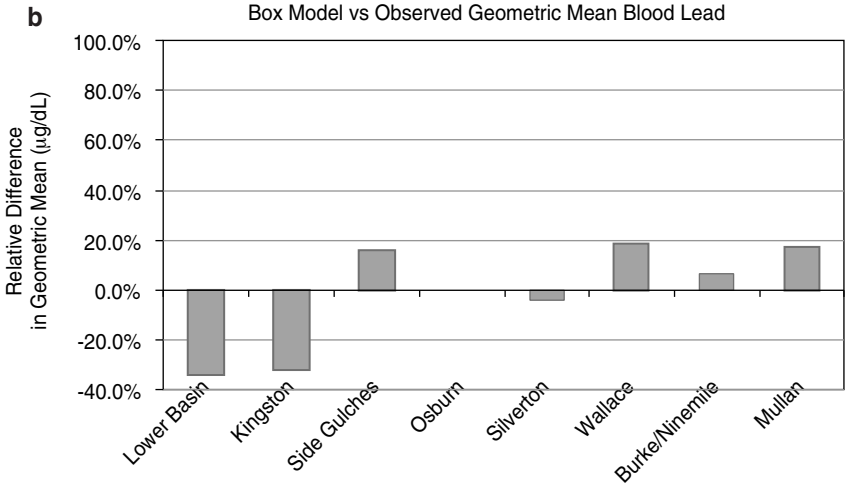

FIGURE 6-8b Relative differences (as percent) between batch mode IEUBK box model prediction and observed geometric mean blood lead (µg/dL) by study area for children 1-5.

slightly, in the lower basin. The box model does a better job of predicting upper basin geometric means but more severely underestimates lower basin values. (It should be noted that relative differences between observations and box model geometric mean predictions in the upper basin are all less than 20%, a relatively small deviation given the current state of modeling of human exposure to environmental contaminants.) The differences between default and box inputs were described previously and are presented in Table 6-4. The box model assumes lower bioavailability and greater contribution of neighborhood soil (as opposed to residential soil and dust) to exposure. Adjustment of bioavailability downward from the default value of 30% is plausible for the upper basin given the observation that the bioavailability of lead from galena is lower than the bioavailability of lead from other minerals in swine feeding trials and that a significant fraction of lead in upper basin soils may be present as unaltered galena (see Table 6-5). However, proportional adjustment of IEUBK results could also be achieved by modifying assumed soil ingesion rates and interpretation of improved model performance acknowledges this uncertainty (von Lindern et al. 2003b) (see Box 6-6 for additional discussion).

It is logical to assume that children may be exposed to lead away from their own residences, but accurate selection of a precise fractional source contribution should not be presumed. Disparate model performance in the lower basin may be related to differing exposure profiles. For example, shoreline recreation in the lower basin may lead to significant exposure to exposed materials with high lead content and bioavailability. Neighborhood soils therefore may be a poor surrogate in the lower basin, leading to box model underprediction. As described in the OU-3 HHRA, follow-up studies of children with high levels of lead in their blood in the lower basin suggest strongly that riverbank material may be an important source of lead exposure (TerraGraphics et al. 2001). The Coeur d'Alene River basin might also exhibit spatial variation in soil lead bioavailability. Smaller particles are transported farther downstream in watersheds and generally exhibit higher lead bioavailability (Mushak 1991) than larger particles.

Adherence/Adequacy of Actions to Superfund Guidelines

Weighting of Biomonitoring Data Versus Model Results

EPA includes two types of IEUBK model calculations in the HHRA, referred to as "community mode" and "batch mode" calculations. Soil lead cleanup levels typically are based on batch mode results, and those results are discussed first here. Batch mode results are a set of predicted BLLs for each individual child in the database for whom "paired data" (soil, dust, and blood lead) are known. At this site, the IEUBK model batch mode

results for the paired data set, using the box model assumptions compare reasonably well with measured BLLs (TerraGraphics et al. 2001, Tables 6-49 and 6-50). For the purposes of the discussion below, the batch mode operation with a paired data set is referred to as step 1. Ideally, the paired data set would be composed of environmental lead levels that are representative of the community; often, it is composed of a biased set of environmental lead levels that do not represent the community at large. In the latter situation, it is clear that if the data set is limited to geographic areas where environmental levels are expected to be high, then the paired BLLs may also be high, and not representative of the community as a whole. However, this is not important for this step because the objective is to explore and understand the relationship between environmental lead and BLLs. To do this, the observed BLLs must be representative of levels that typically would arise upon exposure to these environmental conditions. Good agreement between observation and model predictions is one indication that the observed BLLs are typical of the environmental conditions.

Because the batch mode predictions of BLLs based on environmental lead levels for the paired data set are reasonably good, the next step in the HHRA is to apply the batch mode calculation to all residences and yards in the community for which environmental lead concentrations are available. This is referred to below for purposes of this discussion as step 2. This calculation is done regardless of whether a BLL has been obtained for any child living in the residence. This step produces a predicted distribution of BLLs for the community. If BLLs have been measured for a truly representative cross section of the community (with regard to environmental lead), then the predicted and observed BLLs may be comparable. However, if the measured BLLs (from step 1) are not representative of the distribution of environmental lead levels in the community, it is not appropriate to compare this predicted distribution of BLLs with the observed distribution of BLLs in the community. If the comparison is done and the results are favorable, this suggests that the observed BLLs are a representative cross section of those in the community. However, if the comparison yields unfavorable results, it could be either because the IEUBK model does not work well in this situation or because the observed BLLs are not representative of the community. For example, if the original paired data set used in step 1 included only children who lived in the residences with the highest environmental levels of lead, then when the IEUBK model batch mode is applied to all residences, including those with lower environmental levels, we would expect the overall predicted distribution of BLLs to be lower than the observed distribution. This discussion is presented to demonstrate that blood lead data need not always be "representative" to be useful. However, blood lead data without accompanying environmental lead levels are rarely useful in the modeling context.

TABLE 6-5 EPA Region 8 In Vivo (Juvenile Swine) Studies of Lead Bioavailability in Various Contaminated Soils and Mine Waste Residuals

Site	Sample	Lead (ppm)
New Jersey zinc, Palmerton, PA	Site soil location 2	3,230
	Site soil location 4	2,150
Smuggler Mountain, Aspen, CO	Berm soil	14,200
	Residential soil	3,870
Oronogo-Duenweg mining belt, Jasper County, MO	Near-smelter high-lead soil	10,800
	Near-mill high-lead soil	6,940
	Low-level yard soil	4,050
Murray smelter, Murray City, UT	Slag	11,500
	Surface soil	3,200
Kennecott, Salt Lake City, UT	Residential soil	1,590
	Creek channel material	6,330
Silver Bow/Butte area, Butte, MT	Waste rock dump soils	8,600
Midvale slag, Midvale, UT	Slag	7,895
California gulch, Leadville, CO	Residential soil	7,510
	Trailer park soil	4,320
	Smelter slag	10,600
	Tailings	1,270
N/A	Unweathered crystalline galena in low-lead CO soil	11,200
N/A	NIST powdered leaded indoor paint in low-lead CO soil	8,350

Note: Data shown are lead concentration in material fed, percent of lead mass derived from the most abundant lead mineral and from galena (lead sulfide), particle size range, and the resulting estimated absolute bioavailability (ABA) of lead.
SOURCE: Casteel et al. 1996a-d, 1997a,b, 1998a-e.

BOX 6-6 Are the Assumptions of the Box Model Necessarily Correct?

- The IEUBK box model configuration provides appropriate soil cleanup levels for the Coeur d'Alene River basin OU-3 as a whole.
- Adjusting some of the IEUBK model default values to box model conditions provided a better fit between observed and predicted blood lead values for some but not all geographic subregions of OU-3. Adjustments were based on empirical results, not on knowledge of which parameters more accurately reflect the true state of nature.
- Although such agreement could have been accomplished by reducing the soil/dust ingestion rates, or by lowering specifications for soil/dust bioavailability, the latter option has a more plausible connection to possible geographic differences within the basin. Ingestion rates would not be expected to show patterns of spatial variability.

Mineralogy (as lead mass)	Particle Size (μm)	Suggested ABA (%)
66% manganese-lead oxide, 0% lead sulfide	≤250	34
66% manganese-lead oxide, 0% lead sulfide	≤250	27
62% lead carbonate, 12% lead sulfide	≤250	30
64% lead carbonate, 17% lead sulfide	≤150	31
32% lead carbonate, 0% lead sulfide	≤250	29
57% lead carbonate, 3% lead sulfide	≤250	40
81% lead carbonate, 8% lead sulfide	≤250	40
69% lead oxide, 9% lead sulfide	≤250	27
29% lead-arsenic oxide, 20% lead sulfide	≤250	36
50% lead phosphate, 0% lead sulfide	≤150	15
59% lead or iron-lead sulfate, 9% lead sulfide	≤150	14
36% lead sulfate, 13% lead sulfide	≤250	10
33% lead-arsenic oxide, 6% lead sulfide	≤250	8
≈ 30% lead phosphate, <5% lead sulfide	≤250	37
>70% manganese-lead oxide, 0% lead sulfide	≤250	45
>50% iron-lead oxide, <5% lead sulfide	≤150	9
100% lead sulfide	≤50	3
100% lead sulfide	≤100	<0.5
55% lead carbonate, 0% lead sulfide	N/R	40

Soil lead cleanup levels are derived on the basis of the IEUBK model used for both steps 1 and 2 above. Note that this is actually the same model in steps 1, and 2; in step 1, it is applied only to residences where a child with a blood lead measurement lives, whereas in step 2 it is applied to all residences where environmental measurements have been made. EPA's general approach to calculating a soil lead cleanup level does not need step 2. Rather, it uses the model as applied in step 1 and calculates the highest soil lead concentration that is still consistent with a BLL that, combined with the blood lead GSD, will produce no more than a 5% probability of being above 10 μg/dL. The Coeur d'Alene HHRA takes a somewhat broader, although nearly equivalent, approach, selecting a possible soil lead cleanup level, rerunning the step 2 batch mode run, and considering the predicted blood lead exceedance rate for the residences with soil lead levels within 200 mg/kg of the possible soil lead cleanup level. This approach is some-

what less conservative than the typical approach (it will yield a higher soil lead cleanup level) because the distribution of BLLs predicted for the highest soil lead yards (within 200 mg/kg of the cleanup level) will be slightly lower than predicted for the highest soil lead yard alone. However, the resulting soil lead cleanup level is very similar.

Lack of Site-Specific Bioavailability Assessments

It is well established that some fraction of lead found in soils is absorbable in mammalian gastrointestinal tracts. The absorption of lead from soils from contaminated locales has been studied in juvenile swine by EPA personnel and collaborators (Casteel et al. 1996a-d, 1997a,b, 1998a-e). Findings from these studies are summarized in Table 6-5. Absorption of lead from soil has also been studied in rats (Freeman et al. 1992, 1994, 1996; Dieter et al. 1993). Rats are considered an inferior surrogate for humans, but those data do support trends observed in the swine studies with respect to dependence of availability on speciation. Simulated gastric dissolution of lead-bearing materials has also been conducted in vitro. The results of these studies are generally consistent in demonstrating that a nonnegligible fraction of lead in soil can be absorbed but that efficiency of absorption depends on multiple factors including chemical speciation of lead, dietary factors, and the particle size of soil ingested. Typically, paint-derived lead (lead oxides, basic carbonates) is relatively bioavailable, whereas lead associated with sulfide minerals is relatively unavailable. One study was conducted on soil from a residence within the Bunker Hill box (but not the basin) in human volunteers using a stable isotope approach (Maddaloni et al. 1998). These experiments demonstrated 26% bioavailability of lead in soil to fasted individuals and 2.5% in individuals who consumed lead contaminated soil just after eating.

Given the rather large range of absolute bioavailability (in swine) for soils and residues at site affected by mine waste (Table 6-5), the lack of any such study results applicable to the Coeur d'Alene River basin Superfund site represents a deficiency in the HHRA and the subsequent ROD. A variety of in vivo assays (Freeman et al. 1994; Casteel et al. 1997b) could have been applied; alternatively, an in vitro physiologically based extraction test (Ruby et al. 1996) would have been useful. As demonstrated by Watt et al. (1993), with actual hand wipes from children, the physicochemical form of environmental lead is extremely important in the exposure dynamic. Furthermore, these properties can change over time (Johnson and Hunt 1995), and, because particle size is also important for bioavailability, at a minimum the RI/FS and HHRA ought to have included information on the concentration of lead in different size fractions of basin soils, although EPA guidance does not currently require this. EPA should require that the

IEUBK model used for determining cleanup levels be supported by site-specific measures of bioavailability.

Evaluation/Improvement of Actions Taken for the ROD

Although the committee did not find technical or policy issues with respect to the actions taken for the ROD, in a number of instances science and policy might be considered as conflicting. This is partially a result of the size and complexity of the Coeur d'Alene River basin Superfund site and partly due to advances in scientific knowledge that have not been incorporated into the use of the IEUBK model. We outline a number of examples below.

IEUBK Model Execution Modes

The community-mode IEUBK model runs are not useful because they predict BLLs for the entire community on the basis of mean and range of soil lead levels, but they can be compared only with the subset of BLLs that were measured. If the measured BLLs correspond to children who represent a cross section of environmental lead levels in the community, then this comparison may be adequate. The comparison is shown in Table 6-47 of the HHRA, with mixed results, suggesting that the measured BLLs were not representative of the community (the range of environmental lead conditions used in the model), as discussed in Chapter 5. An alternative explanation is that the IEUBK model does not work well in this situation, possibly because bioavailability may vary from one community to another.

So, what defines a blood lead data set that is useful with the IEUBK model? The HHRA also presents calculations of soil lead cleanup levels following the community mode approach. However, EPA generally does not use this approach in setting soil lead cleanup levels, and it is not consistent with EPA's target for blood lead protection (a target that an individual child have no more than a 5% probability of a blood lead exceeding 10 μg/dL). If this approach were used as a matter of EPA policy to set the soil lead cleanup level, then the representative nature of the BLLs for the community would be a much more important concern. When the batch mode approach is used, as it generally is, and when EPA's individual target for blood lead protection is used, as it typically is, then the blood lead data need not be representative of the community but rather must be representative of the exposures that arise for the observed environmental lead levels. This concept is not articulated in any EPA guidance documents, and clarification is needed; the usefulness of nonrepresentative epidemiological blood lead data may be counterintuitive for scientists and community members alike.

Protection of the Community or of the Individual Child

It appears that it has always been EPA's policy to focus protection on the individual child, but this policy either was not applied or was incorrectly applied at past sites when the community blood lead protection goal was used instead. The community goal, which focuses on keeping 95% of children in a community with BLLs below 10 μg/dL, effectively abandons the 5% of children with BLLs above 10 μg/dL.

Adequacy of the Blood Lead Data

It is the case here that the blood lead screening rate for the community (<30%) is less than EPA often requires at other sites to feel comfortable that a representative cross section of a community has been obtained. EPA makes no decisions based on the predicted or actual average blood lead in the community. So for EPA's purposes, the question is, are the data representative of the BLLs that typically would arise in this community in children who live in houses with the observed environmental conditions (soil and dust lead levels)? This question is key because the IEUBK model and EPA's approach rely on developing an understanding of the relationship between lead in soil and dust and lead in blood. There is no way to answer this question, but there is also no reason to suspect either a systematic high or systematic low bias to the BLLs for these children exposed to their particular environmental conditions. It is possible that there was a community bias in the blood lead sampling toward children with higher BLLs. Presumably, these children live in conditions where they are exposed to higher levels of lead in soil and dust; nutritionally deprived children may be more likely to reside in housing with contamination. However, the soil lead cleanup level is based not on the number or percent of children with elevated BLLs, but only on the relationship between lead in soil and dust and blood lead. Therefore, this community bias, if it exists, does not affect calculation of the soil lead cleanup level.

Compilation of the Blood Lead Data Set

The blood lead data used for comparison in the IEUBK model contained more than one measurement for some children. This has the potential to bias community statistics or the mean and range of blood lead in the community. However, EPA does not use these community statistics in calculating the soil lead cleanup level, so this bias has no effect on selection of the soil lead cleanup level. To the extent that the soil lead cleanup level is based on the results of the HHRA, it is based on the results of the IEUBK model batch mode runs. The batch mode run of the model yields blood lead

predictions for each entry in the data set that is complete or that (at least) contains environmental and blood lead information. If a child is entered in the data set twice by virtue of having a repeat blood lead measurement, then the same environmental lead levels will be used in multiple predictions for this child, and one of the predictions will be closer to observation than the others. Thus comparison or calibration of the IEUBK to site-specific conditions relies on the children sampled being representative of the relationship between blood lead and environmental lead (not on their BLLs being representative of the community). If a child is entered in the data set twice by virtue of having a repeat blood lead measurement, then the same environmental lead levels will be used in multiple predictions for this child, and one of the predictions will be closer to observation than the others. A further bias in comparison or calibration could therefore arise if the children entered in the dataset twice are not representative of the site-specific relationship between blood lead and environmental lead. There is no evidence for either type of such non-representativeness, and any such biases appear likely to be relatively small.

Improvements to Lead Source Apportionment

In Appendix E, it is noted that in perhaps half the houses studied comparing lead, iron and manganese, internal sources for lead in the vacuum cleaner dusts were indicated. Additional studies are needed to confirm this result using other crustal soil tracers and sieve sizes to more accurately characterize the indoor and outdoor sources of lead. Although this analysis was exploratory in nature, it does indicate that there is a value in designing future sampling and analysis programs so that they explicitly address crustal elements concurrently with lead to provide diagnostic information for interpreting the sampling results for lead.

Fortunately, existing sampling protocols involving entryway mats and vacuum bags can provide the analytical results needed to quantify the indoor and outdoor sources of lead in house dust. Specifically, the concentration of lead in indoor dust that is attributable to nonsoil, indoor sources (denoted here as C_{in}) can be estimated by subtracting the concentration of soil-derived lead (C_{sd}) in house dust from the concentration of lead in bulk house dust (C_{bhd}) collected from a vacuum bag. The value of C_{sd} is simply calculated as the product of the dilution ratio and the concentration of tracked-in lead in mat dust (C_t). For example, if the values of C_t and C_{bhd} at a residence are 1,000 and 550 mg/kg, respectively, and the dilution ratio is 0.5 (determined by crustal tracer measurements), then the value of C_{sd} is 500 mg/kg, and therefore C_{in} equals 50 mg/kg. In this particular case, lead from outdoor soil dominates the lead content of the indoor dust. With sufficient samples, the IEUBK model can be run with estimated indoor-outdoor source concentra-

tions for lead in house dust (C_{sd} and C_{in}) to examine how nonsoil sources of lead in dust contribute to BLLs in children. The HHRA, in contrast, conducted statistical analyses between measured BLLs in children and measurements of lead in soils/dusts as well as x-ray fluorescence measurements of lead paint in the children's houses. Those analyses did detect an effect of lead paint on BLLs, but it was small. Unfortunately, x-ray fluorescence measurements are only a surrogate for potential paint-derived lead in house dust, and so it is unclear how representative the results truly are.

Limitations in the Use of Lead Dust Concentrations

An important point to emphasize here is that human activities are the primary source of the dilution effect on substances derived from outdoor soil that have no significant indoor sources. Accordingly, there will be an additional source of variability in the concentrations of lead and other soil-derived substances in dust beyond the variability in outdoor levels. Moreover, the loading of dust on floor surfaces that children come in contact with via hand-to-mouth behaviors is also a function of human activities including the number of household residents, and cleaning frequency, and so forth. Numerous studies have shown that dust lead loading correlates more strongly with blood lead than does dust lead concentration (Aschengrau et al. 1998; Lanphear et al. 1998; Kranz et al. 2004). The IEUBK model, however, determines intakes only as the product of the concentration of lead in soil/dust and an age-adjusted soil/dust ingestion rate prorated for the respective contact media. In essence, the fixed soil/dust ingestion rate used in the IEUBK model is an aggregate parameter that does not take into account variations in house dust loadings that contribute to ingestion exposures. Thus, according to the IEUBK exposure formulation, children in two different houses that have the same concentrations of lead in dust will also have identical lead ingestion exposures, even though the loadings of dust and lead on indoor surfaces of the houses could vary substantially.

Atmospheric Lead Contributions to Indoor Dust Exposure

To assess the potential significance of airborne lead levels on surface loadings indoors, we prepared a screening-level analysis of the inputs of lead to floor surfaces from footwear tracking and deposition of suspended particles derived from the infiltration of outdoor particles through a building shell. Table 6-17 of the HHRA provides data on the fluxes of lead into houses situated in several Coeur d'Alene River basin communities. The geometric mean values range from 0.48 mg/m^2/day (in the lower basin/Caltaldo) to 4.28 mg/m^2/day (for Burke/Ninemile). These flux values, however, are only for the entryway mats—not floors in the interior of the

houses sampled. Equivalent floor loading rates due to lead redistribution by foot traffic can be estimated by multiplying mat loading rates by the mat area (0.318 m^2; von Lindern et al. 2003b) to obtain a whole-house mass-loading rate (in mg/day) that is then divided by an effective house floor area. For a lead mat loading rate of 1 mg/m^2/day and an assumed floor area of 100 m^2 (about 1,000 square feet), the resulting lead floor loading rate is about 3 $\mu g/m^2$/day.

The atmospheric deposition rate onto floor surfaces can be calculated as the product of a particle settling velocity and an indoor air concentration of lead. With a reference outdoor lead concentration of 0.10 $\mu g/m^3$ and an indoor level 0.03 $\mu g/m^3$ (based on the IEUBK default indoor/outdoor value of 0.3), the associated loading rates would be 0.18 and 1.5 $\mu g/m^2$/day, respectively, for gravitational settling velocities of 0.25 and 2.1 m/hour, based on outdoor-derived particles 1 and 3 m in diameter (Milford and Davidson 1985) and a density of 2 g/cm^3. These values would represent between 5% and 32% of the total flux from both foot traffic and surface deposition. The composite concentration of lead in dust resulting from tracked-in soils on floors and deposition will vary according to the amount of particulate matter introduced by the various transport processes and indoor sources as well as other indoor lead sources. Given that the levels of lead in ambient air would have been much higher within the box when the box version of the IEUBK model was initially being developed, it is conceivable that the community soil parameter is actually a surrogate parameter that represents airborne lead derived from soil resuspension.

The IEUBK model predicts that 10 μg of lead per gram of dust would be attributable to atmospheric lead at its default concentration (0.1 $\mu g/m^3$)—based on a simple ratio of the concentration of lead indoor dust to the level in outdoor air. Unfortunately, house dust is associated with many indoor surfaces, including nonfloor horizontal surfaces such as sofas, chairs, tables, beds, and the concentrations of lead in the associated dust loadings will vary, as will ingestion exposures related to hand-to-mouth contacts with those surfaces. In essence, the IEUBK exposure module is really an oversimplification of the transport and fate processes that control indoor lead, and it is time that more mechanistically based approaches are adopted so that the exposure component of the IEUBK model is commensurate with the lead biokinetic module.

CONCLUSIONS AND RECOMMENDATIONS

In this section, the committee provides several conclusions and recommendations regarding the application of the IEUBK model in the basin and general comments on model use, function, and associated EPA guidance. This section is intended to facilitate the development of the model as a

scientific tool for more accurately assessing expected children's blood lead concentrations and support the model's future application at sites with lead contaminated soil. As provided in the statement of task (Appendix A), "the committee will strive to provide guidance to facilitate scientifically based and timely decision making for this site in the future." As such, the conclusions and recommendations herein are intended to guide future decision making and not to elicit a reconsideration of the ROD for the Coeur d'Alene River basin.

Conclusion 1

Multicompartment predictive blood lead models are powerful tools for pediatric lead-exposure risk assessments, for exploring lead risk management options, and for crafting remediation strategies. Their application to Superfund sites with environmental lead contamination is an important part of the CERCLA regulatory process.

Conclusion 2

Design and functioning of the IEUBK blood lead prediction model are consistent with current scientific knowledge, but improvements could be made. Specifically, substantial unaddressed uncertainty exists in three areas: model computations, input parameter values, and application of model computations to populations of individuals.

These uncertainties are discussed in this chapter and are summarized as follows: (a) Errors and inconsistencies exist in the documentation and computer code used for model implementation, as defined in this chapter and detailed in Appendix C. (b) Uncertainties in the input parameters of bioavailability and soil/dust ingestion rate can lead to significant variations in model predictions, as illustrated in Table 6-3. Although site-specific measures of bioavailability can be made, measuring ingestion rate parameters is far more difficult and there is little agreement on their measures of CT. Difficulty in making ingestion rate measurements suggests that many (if not most) model users will employ the model default values; these have not been reevaluated for more than 12 years. (c) Point estimates are projected to population distributions by making assumptions; application of a default probability density function parameter to a point estimate is not a proper way to define a population. Probabilistic exposure modules interfaced with the IEUBK biokinetic computations have been produced (for example, integrated stochastic exposure; [SRC 2003]) and could be subjected to the same validation and verification used for the IEUBK. These approaches would provide a more scientifically sound basis to project risk calculations for populations of individuals.

Recommendation 1

After correcting errors, EPA should recompile the IEUBK model source code using state-of-the-art algorithms for integration. Cornerstones of this program should be open access to the source code for the IEUBK model and any subsequent probabilistic exposure model implementation versions of it and a peer review process to ensure its accuracy.

Recommendation 2

EPA should undertake a significant effort to improve the knowledge base for soil/dust ingestion rates. Effort in this area will bring benefits for many other contaminant-exposure risk assessments for which soil ingestion is a significant exposure pathway.

Recommendation 3

EPA should proceed with implementating a probabilistic, stochastic exposure model version of the IEUBK and initiate the verification and validation process for it. This would substantially end the debate about application of default or site-specific GSD values for model use in establishing cleanup levels. In the interim, the agency should establish a comprehensive, uniform policy for use of site-specific GSD values to be utilized in model computations and should promulgate guidelines for its determination.

Conclusion 3

The IEUBK model was adequately and appropriately used in the Coeur d'Alene River basin, although the optimum application was not undertaken. Most importantly, site-specific bioavailability would have improved the application of the model, and better characterization of the physicochemical properties of the exposure materials would have enhanced the credibility of the results.

Conducting IEUBK model evaluations using solely default parameters, without their justification, has little utility because risk assessments should not be based on default parameters. The box model incorporated in both the HHRA and the ROD used a deviation from the IEUBK model default values for bioavailability. Given the wide range of values reported at other sites affected by mining (Table 6-5), it would seem that measurements of bioavailability in the Coeur d'Alene River basin should have been carried out. Furthermore, since natural soil processes can lead to alteration of mineral forms and conceivably either increased or decreased bioavailability over time, the likelihood and consequences of such changes should have been discussed.

At the very least, estimates of the lead-exposure impact would have been improved by determination of the lead concentrations in various soil particle size fractions. Such results would have improved interpretation of soil transport from outdoor to indoor environments. If the EPA had used their bulk analyses for the crustal elements iron and manganese as the committee did, a better justification would have evolved for the structure of the box model extension to the rest of the Coeur d'Alene River basin.

Recommendation

EPA should require that IEUBK model use for determining cleanup levels be supported by site-specific measures of bioavailability and that particle-size-range lead concentration determinations be undertaken. Increased emphasis should be placed on acquiring analytical metrics that quantify the strength of the lead-based paint source(s). In addition, EPA should emphasize the interpretive benefits for source attribution that derive from additional soil and dust bulk chemical measures (for example, aluminum, silicon, iron, manganese, and calcium) and encourage acquisition of such data where feasible. EPA should consider that ingestion rates might be site specific and undertake fundamental research aimed at addressing this hypothesis.

Conclusion 4

Alternative tools for assessing the validity of model predictions were underutilized in interpretations of model results. For example, other models were not used in the assessment. The committee's analysis of alternative models suggests that at this site the outcome of additional analyses would not have affected remedial decisions, but, had they been used as part of the HHRA for inclusion in the ROD, the scientific credibility of the decisions reached would have been enhanced.

Not using alternative analyses resulted in the loss of opportunity for expanding the scientific knowledge associated with application of predictive models to real world situations. Although some alternative interpretive tools were used in the development of an IEUBK model prediction regime, such as the structural equation modeling for the regression analyses in the HHRA, use of additional techniques would have helped solidify application of the box model as it was eventually constructed. For instance, the collection of mat dust lead (and other metal) concentrations and loading rates proved to be valuable additions to the RI/FS protocols. Appropriate analysis of the iron and manganese data would have provided additional supporting evidence upon which to base a soil contribution of 60% for indoor dusts. Similarly, a comparison of box model predictions by the IEUBK and

the O'Flaherty models, showing identical cleanup-level determinations, would have highlighted the critical importance of uncertainty in bioavailability and ingestion rate parameters.

Recommendation

EPA should promote use and development of both deterministic and probabilistic multipathway uptake and pharmacokinetic models for lead as research tools and provide scientific maintenance for their continued development and improvement. This could substantially improve their application as regulatory instruments.

Conclusion 5

The committee finds that EPA guidance concerning specific use of the IEUBK model and additional use of blood lead studies is incomplete. The inherent uncertainties associated with model predictions coupled with the high value placed on the need for predictive capability in the protection of both present and future populations requires a more clear and comprehensive articulation of IEUBK model-use policy.

The 1998 OSWER directive fails, as described in this chapter, to give adequate guidance about what to do when BLLs and IEUBK model results disagree by a substantial margin. It states without clear justification that model results are to take precedence in these situations. Significant emphasis in the directive suggests that, where such disagreement exists, the blood lead study may be suspect. It is clear that blood lead observations may not always be representative of the population, may have been conducted at the wrong time of year, or may have been influenced by significant knowledge of lead hazards within a population. However, uncertainties may also exist in the IEUBK model results, where the relationship between soil and dust may not be well understood, the bioavailability of soil and/or dust may be unknown, or where factors, such as lead in paint, may be inadequately addressed in the model input parameter characterizations. Additional information for addressing such uncertainties could be provided by assays of soil and dust bioavailability, determining the presence or absence of lead-based paint, which can serve as a confounder in the model, and by analyses of additional metals such as arsenic, cadmium, and zinc as these metals may co-occur with lead and can improve the estimate of soil transfer to dust.

Recommendation

EPA's guidance on use of blood lead studies in conjunction with the IEUBK model needs clarification, especially on protocols for reconciling

differences between modeled and observed blood lead values and for objectively considering the uncertainties associated with each. The guidance/policy should address the following points:

- Where blood lead observations are available, a systematic protocol for comparison of predicted and observed BLLs should be used for all risk assessments, and an acceptable level of variability between such results should be established to define "significant" differences.
- Criteria should be established upon which to judge whether or not the extant blood lead observations are representative of the community concerned, covering the full range of lead-exposure potential. If "significant" differences exist between observed and predicted blood lead values, such criteria would establish whether an additional blood lead study effort was required.
- Definitive guidelines for the conduct of blood lead studies should be established. The focus should be on the coherence of the joint data set covering the full range of lead exposure risks and the collection of blood lead data associated with that range of exposure.
- When model results and acceptable blood lead study observations do not agree, and when default IEUBK exposure values have been used for some or all of the modeling exercise, additional information should be collected to examine uncertainty in model inputs and to ensure that all exposure sources and lead uptake/intake rates have been adequately established for the specific site in question.
- Before development of a fully probabilistic IEUBK model, uncertainty in the GSD should be explored with the ISE, lead risk model, or another similar model to understand how it may depart from the default for a particular site.

Conclusion 6

The IEUBK model results should not be the sole criterion for establishing health-protective soil concentrations at mining megasites such as OU-3 of the Coeur d'Alene River basin, because model uncertainty and site complexity may interact in unexpected or unknown ways.

This chapter details a variety of specific challenges associated with IEUBK application to OU-3. The geographic area defined as OU-3 exhibits a great diversity of topography, land use practice, bedrock geology, ecologic community structure, and hydrologic regime. Consequently, one would expect the nature and extent of natural geochemical mineral alteration, soil digenetic processes, and sediment transport and deposition dynamics to vary accordingly. Such variations are manifest in the IEUBK box model predictions, which suggest regional differences between the upper

and lower basin in lead bioavailability and possibly in other model operation parameters as well. By extension, it is likely that similar problems will arise at other sites where ecologic, geomorphological, and sociodemographic complexity of this nature exists. A comprehensive revision of the 1998 OSWER directive on model use, incorporating those issues just outlined, is needed to adequately address issues associated with geographic variability at large geographically heterogeneous sites.

Recommendation

Incorporate the IEUBK model in a negotiated and carefully communicated HHRA/ROD structure for which the primary prevention paradigm contains the four fundamental elements of

- Predictive capability (IEUBK or successors)
- Empirical results (blood lead study results)
- Economic feasibility
- Sustainable remediation (long-term remedy maintenance)

Each of these key elements is necessary for successful remediation, but the way they are weighted for the mutual satisfaction of all stakeholders may be different across the variety of contiguous spatial elements defined for the OU. Both risk assessment and risk management activities should be structured according to natural environmental system boundaries; they should not represent the aggregation of apparently applicable policies previously found to be successful for smaller, simpler systems.

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